
Potential Protective Roles of Tamarind Fruit Pulp Aqueous Extract Against Hyperglycemic and Testicular Toxicity in Alloxan Induced-Diabetic Male Rats

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Abstract

The present study aimed to evaluate the potentially protective effects of aqueous extract of tamarind fruit pulp (AETF) against hyperglycemia and male infertility in diabetic rats. AETF was getting by soaking tamarind fruit pulp in distilled water in a ratio of 1:2 (fruit pulp: water) for 12 hr. After soaking period, the solution heating for 20 min at 50° C, then filtered and concentrated under vacuum. In the present study, thirty-five male albino rats were used and divided into five groups (each of= 7 rats). Group 1 served as normal rats, group 2 served as untreated diabetic rats; and diabetic groups 3, 4, and 5 were given orally concentrated AETF at a dose of 1,2 and 3ml/100g body weight/day, respectively. The results revealed that, oral administration of AETF for diabetic rats significantly ($P < 0.05$) improved testes, seminal vesicles and prostate glands weight, compared with untreated diabetic rats. The treated diabetic rats had a significant lower ($P < 0.05$) in BGL, serum activities of AST, ALT and ALP enzymes; and larger in serum insulin, testosterone, LH and FSH levels, compared to untreated diabetic rats. In the testes tissues, AETF significantly ameliorated MDA level and activities of GSH, GPx, SOD and CAT enzymes in diabetic rats. Histopathological investigation for testes of tread dia-

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betic rats showed almost normal histological structure of seminiferous tubules with the lumen filled of spermatozoa, especially with higher level of AETF. In conclusion, AETF exhibited hypoglycemia, antioxidant activity and protective effect against male infertility in diabetic rats.

Keywords: Tamarind fruit; Antioxidant enzymes; Sex hormones; Diabetes Mellitus; Histopathological

Introduction

Tamarindus indica Linn (family: *Caesalpinaceae*) fruit is sweet and sour in taste and known as tamarind and is a known as medicinal plant. Tamarind pulp has been declared to contain citric, tartaric, ascorbic and maleic acids; and β -carotene (**Roy et al., 2010**). In Indian, traditional medical system prescribes tamarind fruits for constipation, indigestion and flatulence, and the seeds are prescribed for diabetes, while in the Bangladesh medicinal practitioners, both seeds and fruits are prescribed for diabetes (**Havinga et al., 2010**). Furthermore, tamarind fruits have been shown to have antioxidant, anti-inflammatory, antibacterial and anti-diabetic properties as well as hypolipidaemic and hepatoprotective effects (**Wagh and Bhagure, 2012**).

Diabetes mellitus is one of the fundamental chronic endocrine disorders characterized by hyperglycemia and defects in the metabolism of macronutrients (carbohydrates, proteins and fats). Oxidative stress evolves complications in diabetic (both types 1 and 2) and engages in beta cell destruction in type 2 diabetes. However, controlling blood glucose levels, plays an important role in maintaining the antioxidant and pro-oxidant balance (**Nahar et al., 2014 and Hatanaka et al., 2016**).

Alloxan is used to induce diabetes mellitus to screen antidiabetic effect of some plants and herb materials in experimental animals. It has been found to induce diabetes by damaging pancreatic islet cells by liberate free radicals, which in turn involved in damage the several tissues which occurs in the progression of diabetes disease (**Prakash et al., 2010**).

Normally, there is a balance between antioxidant activities and the production of reactive oxygen species (ROS) in the male reproductive organs. Oxidative stress induced imbalance between production of reactive oxygen species and antioxidant activity becomes the interesting focus as the potency cause of male infertility (**Vijay et al., 2012**). Recently, the use of natural plant and herbal medicines has become more predominant in the treatment of many diseases. Hence, the present study was conducted to evaluate the potentially protective effects of aqueous extract of tamarind fruit pulp (AETF) against hyperglycemia and male infertility in diabetic rats.

Materials and Methods

Materials

Tamarind Fruit: Tamarind fruit was purchased from Harraz company for Agricultural seeds and medicinal plants, Cairo, Egypt. Tamarind fruit was taxonomically and identified by the experts of the National Center for Agricultural Research, Giza, Egypt.

Male Rats: Thirty-five male albino rats Sprague-Dawley strain, aged (10-12) weeks and weighing 185 ± 5 g which were bred in animal house, National Cancer Institute, Cairo University, Egypt, were used in the present experiment.

Basal Diet: Casein, cellulose, choline chloride, D-L methionine, vitamins and minerals mixture constituents were purchased from El-Gomhouria Company for Trading Drugs, Chemicals and Medical Instruments, Cairo, Egypt. Starch, soybean oil, and sucrose were obtained from the local market, Cairo, Egypt.

Chemicals and Kits: Alloxan, kits for biochemical analysis and others chemicals were purchased from the Gamma Trade Company for Pharmaceutical and Chemical, Dokki, Egypt.

Methods

Preparation of Tamarind Fruit Pulp Extract: The aqueous extract of the tamarind fruit pulp (AETF) was achieved as described by **Pattar *et al.*, (2013)** with some modifications. In brief, fruit pulp was soaked in distilled water in the ratio of 1:2 (pulp: water) for 24 hr at refrigeration temperature (5° C). After the soaked period, the solution heating at 50° C for 20 min and handily homogenized and filtered through cheesecloth into glass container. Then, the filtered solution was concentrated under vacuum to obtained on concentrated extract and stored to carry out the further process.

Preparation of Basal Diet: The basal diet (AIN-93M) was formulated according to **Reeves *et al.*, (1993)** to meet the recommended nutrients level for rats. It consists of casein (20%), vitamins mixture (1.0%), minerals mixture (4.0%), soybean oil (5%), fibers (5%), L-Cystine (0.18%) Choline chloride (0.20%), sucrose 10% and the reminder was corn starch.

Induction of Diabetes and Experimental Design: Diabetes was induced by subcutaneous injection using a single dose of alloxan dissolved in sterile normal *saline* at a dose of 150 mg/kg of body

weight as stated by **Buko et al., (1996)**. Diabetic rats were kept for the next 24 hr on 10% glucose solution to prevent hypoglycemia. After seventy-two hours of alloxan injection and after fasting rats for 4-hrs, blood glucose levels were measured from the tail vein. Rats with a blood glucose level from ≥ 300 mg/dl were considered diabetic and included in the present experiment. Then, all rats were divided into five groups (each of= 7 rats). The experimental groups were as follows: the first group served as normal rats (negative group) and were injected with an equivalent amount of saline solution. The second group served as untreated diabetic rats (positive group) and received distilled water orally. Diabetic groups (3), (4) and (5) were orally received 1, 2 and 3ml/100g body weight/day of concentrated AETF. All groups of animals were kept in a healthy animal house at 20-25 °C, 12:12 hr light/dark cycle, fed on the basal diet, and water were provided *ad libitum* for four weeks.

At the end of the experimental period (4 weeks), all rats were fasted for 12 hr and each rat was weighed. Blood samples were collected from the portal vein into dry-clean centrifuge tubes. Serum samples were separated by centrifuge at 3000 r.p.m for 15 min and obtained serum were used for biochemical analysis. Testes, prostate glands and seminal vesicles were accurately removed out from the sacrificed animals and weighed. Then, testes were prepared for tissues analysis and histological examination.

Biochemical Analysis

Determination of Blood glucose and Serum Insulin Levels: At the end of experimental period and after the rats had been fasted for 12 hrs., blood samples were collected by tail vein of the rats and blood glucose levels were measured immediately by using a single touch Glucometer (Ascensia ENTRUST, Bayer). The serum insulin

levels were measured by a sensitive rat insulin radioimmunoassay (RIA) kit (Diamond Co, Hannover, Germany) according to the described method by **Posario, (2010)**.

Determination of Serum AST, ALT and ALP Activities: Activities of serum aspartate aminotransferase (AST) and serum alanine aminotransferase (ALT) and alkaline phosphatase (ALP) enzymes were determined colorimetrically using spectrophotometer as described by **Bergmeyer et al., (1978)** and **Roy, (1970)**, respectively, in the kit's instruction (Diamond Co, Hannover, Germany). The absorption of the test samples was read at 505nm and 510nm for AST, ALT and ALP, respectively.

Determination of Serum Testosterone, LH, FSH Hormones: Serum levels of testosterone, luteinizing hormone (LH) and follicle stimulating hormone (FSH) were determined using ELISA Kits (Monobind Inc. USA) according to the described methods by **Tietz, (1995)** and **Rebar et al., (1982)**, respectively.

Determination of Malondialdehyde and Antioxidant Enzymes of Testes Tissues: One gram of frozen testicular tissue from each rat was washed with ice-cooled NaCl solution (0.9%) and homogenized in 100 ml of ice-cooled potassium chloride (1.5%) and 50 mmol potassium phosphate buffer solutions (pH 7.4) to yield 1% homogenate (W/V). The homogenized testes tissues were centrifuged at 4000 rpm for 10 min at 4°C. The obtained supernatants were maintained in sample bottles, frozen overnight to ensure the maximum release of the antioxidant enzymes as described by **Oyedemi et al., (2011)**. The obtained supernatants were analyzed to determine malondialdehyde (MDA) concentrations and activities

of reduced glutathione (GSH), glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) enzymes using commercial assay kits according to described methods by **Ohkawa *et al.*, (1979)**, **Sedlak and Lindsay, (1968)**, **Paglia and Valentaine (1976)**, **Spitz and Oberley, (1989)** and **Sinha, (1972)**, respectively.

Histopathological Examination: Testes of the scarified rats were taken and flooded in 10% formalin solution. The specified specimens were then trimmed, washed and dehydrated in ascending grades of alcohol. Then, specimens were cleared in xylol, embedded in paraffin, sectioned at 4-6 microns' thickness, and spotted with heamtoxylin and eosin stain for the histopathological examination as described by **Carleton, (1979)**.

Statistical Analysis: Statistical analysis of all the obtained data was completed using one-way analysis of variance (ANOVA) using the Origin Lab (SPSS Ver. 20) software. The obtained results were expressed as mean \pm standard division (mean \pm SD). Significant differences among experimental groups considered at $P < 0.05$.

Results

As shown in Table 1, Diabetes significantly ($P < 0.05$) decreased the weight of testes (2.66 ± 0.10) and seminal vesicles (0.67 ± 0.07), and increased prostate gland weight (0.89 ± 0.07), compared with that of the normal rats (4.87 ± 0.11 , 1.51 ± 0.11 and 0.58 ± 0.53 , respectively). Oral administration of the concentrated AETF in a daily dose (1, 2 and 3 ml/100g of b.wt) to diabetic rats for 4 weeks significantly ($p < 0.05$) prevented the decreases of testes and seminal vesicles weight and the increase in prostate gland weight, compared with that of the positive control rats (untreated diabetic rats).

The effect of oral administration of AETF on BGL and serum insulin levels in diabetic rats is presented in Table 2. Consecutive to alloxan administration, a significant increase of BGL (300.01 ± 1.21 mg/dl) and decrease in serum insulin (0.97 ± 0.11 ng/ml) levels were observed, compared to normal rats (102.86 ± 1.95 mg/dl and 3.12 ± 0.30 ng/ml, respectively). In comparison to the positive control group, the three doses of AETF (1, 2 and 3 ml/100g b.wt/ day) significantly ($p < 0.05$) lowered the increase of BGL and the decrease of serum insulin.

Table 1: Effect of oral administration of AETF on relative weight of sexual organs to body weight in diabetic rats.

Groups	Testes	Seminal vesicles	Prostate glands
Group 1: Normal rats	4.87 ± 0.11^a	1.51 ± 0.11^a	0.58 ± 0.53^d
Group 2: Diabetic rats	2.66 ± 0.10^d	0.67 ± 0.07^d	0.89 ± 0.07^a
Group 3: Diabetic rats + 1 ml/100 g b.wt/day of AETF	2.94 ± 0.05^c	0.90 ± 0.10^c	0.71 ± 0.07^b
Group 4: Diabetic rats + 2 ml/100 g b.wt/day of AETF	3.83 ± 0.53^b	1.33 ± 0.08^b	0.64 ± 0.05^c
Group 5: Diabetic rats + 3 ml/100 g b.wt/day of AETF	4.66 ± 0.18^a	1.47 ± 0.08^a	0.56 ± 0.05^d

Each value represents the mean \pm SD. Means with the different superscript letters in the same column were different significantly at $P < 0.05$

Table 2: Effect of oral administration of AETF on BGL and serum insulin levels in diabetic rats.

Groups	BGL (mg/dl)	Insulin (ng/ml)
Group 1: Normal rats	102.86±1.95 ^e	3.12±0.30 ^a
Group 2: Diabetic rats	300.01±1.21 ^a	0.97±0.11 ^d
Group 3: Diabetic rats + 1 ml/100 g b.wt/day of AETF	273.43±1.90 ^b	1.37±0.11 ^c
Group 4: Diabetic rats + 2 ml/100 g b.wt/day of AETF	188.43±2.76 ^c	2.29±0.17 ^b
Group 5: Diabetic rats + 3 ml/100 g b.wt/day of AETF	107.57±1.40 ^d	3.19±0.15 ^a

Each value represents the mean ± SD. Means with the different superscript letters in the same column were different significantly at P < 0.05

The listed results in Table 3 illustrate the effect of oral administration of AETF on serum activities of AST, ALT and ALP enzymes in diabetic rats. In the untreated diabetic group, the serum activities of AST, ALT and ALP enzymes increases significantly (p < 0.05) compared to a normal control group, but accompanying treatment with the different doses of AETF significantly (p < 0.05) reduced the serum activities of AST, ALT and ALP enzymes, compared to the positive group.

Effect of oral administration of AETF on the level of MDA and activities of antioxidant enzymes (GSH, GPx, SOD and CAT) in testes tissues are shown in Table 4. The presented results revealed that diabetes significantly (P<0.05) increased MDA levels and decreased activities of GSH, GPx, SOD and CAT antioxidant en-

zymes in testicular tissue as compared to that of normal rats. In contrast, treatment with AETF induced a significant decrease in MDA levels and increased activities of antioxidant enzymes (GSH, GPx, SOD and CAT) in testicular tissue as compared to that of untreated diabetic rats.

The effect of oral administration of AETF on the serum sex hormone levels (testosterone, LH, FSH) in diabetic rats is presented in Table 5. Recorded results showed that untreated diabetic rats had significant ($P < 0.05$) decreases in serum testosterone, FSH and LH levels compared with the normal rats. Oral administration of AETF significantly ameliorated serum testosterone, LH and FSH levels when compared to the positive control group (diabetic rats).

Table 3: Effect of oral administration of AETF on serum activities of AST, ALT and ALP enzymes in diabetic rats.

Groups	AST (IU/l)	ALT(IU/l)	ALP(IU/l)
Normal rats	45.14±1.78 ^e	58.43±1.72 ^e	98.57±1.81 ^d
Diabetic rats	107.14±2.34 ^a	119.29±1.50 ^a	172.43±2.76 ^a
Diabetic rats + 1 ml/100 g b.wt/day of AETF	80.71±2.69 ^b	90.57±2.23 ^b	150.14±1.46 ^b
Diabetic rats + 2 ml/100 g b.wt/day of AETF	68.14±1.77 ^c	73.71±2.69 ^c	127.57±1.51 ^c
Diabetic rats + 3 ml/100 g b.wt/day of AETF	50.29±2.87 ^d	60.86±1.46 ^d	100.43±1.27 ^d

Each value represents the mean ± SD. Means with the different superscript letters in the same column were different significantly at $P < 0.05$

Table 4: Effect of oral administration of AETF on the MDA level; and the activities of GSH, GPx, SOD and CAT antioxidant enzymes in testes tissues of diabetic rats.

Groups	MDA nmol/mg tissues	GSH nmol/mg tissues	GPx nmol/mg tissues	SOD U/mg tissues	CAT μmol/mg tissues
Normal rats	1.39±0.09 ^d	37.00±0.82 ^a	25.43±0.98 ^b	19.14±0.69 ^a	312.43±2.07 ^a
Diabetic rats	2.44±0.08 ^a	22.00±1.15 ^d	11.29±0.95 ^e	8.79±0.27 ^d	160.29±1.70 ^d
Diabetic rats + 1 ml/100 g b.wt/day of AETF	1.91±0.09 ^b	25.43±0.53 ^c	16.00±0.82 ^d	13.43±1.13 ^c	280.00±2.31 ^c
Diabetic rats + 2 ml/100 g b.wt/day of AETF	1.63±0.16 ^c	30.43±0.79 ^b	20.71±0.76 ^c	16.00±0.82 ^b	276.14±1.95 ^b
Diabetic rats + 3 ml/100 g b.wt/day of AETF	1.33±0.05 ^d	36.4±0.90 ^a	26.57±0.98 ^a	19.29±0.76 ^a	311.71±1.50 ^a

Each value represents the mean ± SD. Means with the different superscript letters in the same column were different significantly at $P < 0.05$

Table 5: Effect of oral administration of AETF on the serum sex hormone levels (testosterone, LH, FSH) in diabetic rats.

Groups	testosterone ml U/ml	LH ml U/ml	FSH ml U/ml
Normal rats	4.31±0.12 ^a	4.00±0.14 ^a	3.46±0.10 ^a
Diabetic rats	1.26±0.14 ^e	1.67±0.16 ^d	1.70±0.81 ^d
Diabetic rats + 1 ml/100 g b.wt/day of AETF	1.77±0.11 ^d	2.33±0.14 ^c	2.16±0.33 ^c
Diabetic rats + 2 ml/100 g b.wt/day of AETF	2.60±0.12 ^c	2.91±0.10 ^b	2.64±0.10 ^b
Diabetic rats + 3 ml/100 g b.wt/day of AETF	4.07±0.11 ^b	3.87±0.11 ^a	3.16±0.13 ^a

Each value represents the mean ± SD. Means with the different superscript letters in the same column were different significantly at $P < 0.05$.

Histopathological Results

Histopathological structure of testes from normal rats showed no histological changes of mature seminiferous tubules associated with plenary spermatogenic series and the lumen was filled with mature spermatozoa as shown in Photo 1. In contrast, testis sections of untreated diabetic rats (positive group) revealed large vacuolations, degeneration and necrosis of germ cell lining of seminiferous tubules and oligospermia as showed in Photo 2. Microscopically examination for testes of treated diabetic rats with AETF at a dose of 1 ml/100g b.wt/day revealed conservative degeneration and necrosis associated with interstitial edema (Photo 3). Testes of diabetic rats given orally AETF at a dose of 2 ml/100g b.wt/day revealed nearly normal histological architecture with normal spermatogenic series (Photo 4). However, the testes of treated diabetic rats with 3 ml/100g b.wt/day have normal histological structure with normal spermatogenic series and the lumen of seminiferous tubules was filled with spermatozoa as showed in Photo 5.

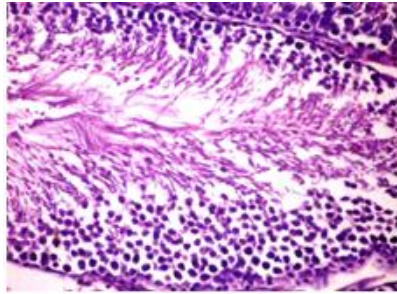


Photo (1): Testis of a normal rat exhibiting normal histological structure of mature functioning seminiferous tubules associated with complete spermatogenic series. (H and E, X 400)

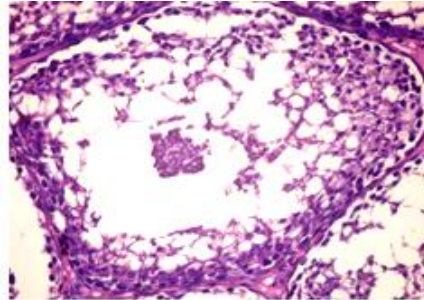


Photo (2): Testis of a diabetic rat appearing large vacuolations, degeneration and necrosis of germ cell lining of seminiferous tubules and oligospermia. (H and E, X 400)

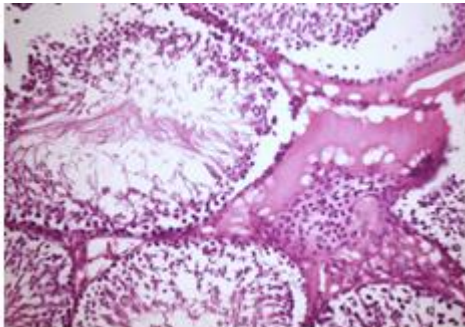


Photo (3): Testis of a diabetic rat given orally AETF in a dose of 1 ml /100 g b.wt/day showing modest degradation of cell germ lining of somniferous tubules associated with interstitial edema. (H and E, X 400)

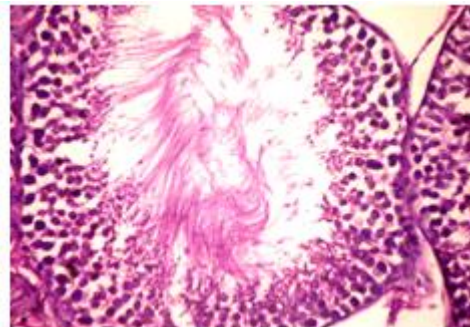


Photo 4: Testis of a diabetic rat given orally AETF in a dose of 2 ml/100 g b.wt/day showing near normal histological structure of seminiferous tubules with the lumen replete with spermatozoa. (H and E, X 400)

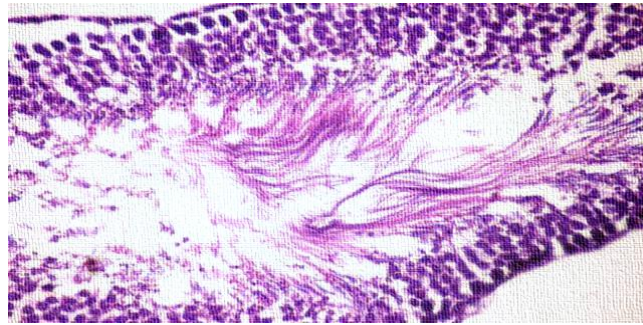


Photo 5: Testis of a diabetic rat given orally AETF in a dose of 3 ml/100 g b.wt/day exhibiting normal histological structure of seminiferous tubules with the lumen filled with spermatozoa. (H and E, X 400)

Discussions

The present study was accomplished to inspect the potentially protective effects of aqueous extract of the tamarind fruit pulp (AETF) against hyperglycemia and male infertility in diabetic rats.

Induction of diabetes by subcutaneous injection using a single dose of alloxan (150 mg/kg of b. wt) caused higher in blood glucose and lower in serum insulin levels; and testicular toxicity and oxidative stress. Testicular toxicity and oxidative stress in diabetic rats were characterized by the significant decreases in weight of the testes and seminal vesicles and increases in prostate gland weight, as well as decreases in serum activities of AST, ALT, ALP enzymes; and levels of sex male hormones (testosterone, FSH and LH). Furthermore, there were lowered in testicular antioxidant capacity; degeneration and necrosis in testicular tissue. The present findings are agreed with **Gaunay *et al.*, (2013)** who indicated that

diabetes is one of the main leading causes of reproductive disorders characterized by testicular tissue dysfunction, lowered testicular weight, quantity of sex cells, sperm quality and sex hormonal disorder, as well as increased the risk of seminiferous tubule degradation. Furthermore, **Asmat *et al.*, (2016)** showed the link between diabetes and oxidative stress as indicated by the higher in lipid peroxidation and the lower in cellular antioxidant defenses, which play an important role in the development of male infertility complications in diabetic patients. Recently, **Shoorei *et al.*, (2019)** and **Sadoughi *et al.*, (2019)** showed significant change in testis tissue structure with germ cell apoptosis in testicular tissue of adult diabetic rats.

The obtained results may be related to oxidative stress, results from the unbalance of endogenous antioxidant systems and reactive oxygen species. In addition to that the spermatozoa possess a weak antioxidant defense system and liable to oxidative stress, which effect on the sperm integrity (**Bisht *et al.*, 2017**). Furthermore, oxidative stress impairs the steroidogenic ability of Leydig cells and the differentiation ability of the germinal epithelium (**Aitken and Roman, 2008**) and has been associated with pathologic aftermaths in the male reproductive organs (**Lee *et al.*, 2019**).

As established in the present study, oral administration of AETF to diabetic rats, in a dose-dependent manner induces significant ameliorated in blood glucose, serum insulin levels, activities of AST, ALT and ALP enzymes, as well as the protective effects against male infertility and oxidative stress. There was also a marked increase in the activity of antioxidant enzymes in testicular tissue and alleviation the testicular degenerative changes. The protective effect of AETF on male fertilizing capacity manifested by the significant increases in weight of male sexual organs and serum

testosterone, LH and FSH levels. This protective effect of AETF against alloxan- induced diabetic and testicular injury was partially similar to the obtained results by **Roy *et al.*, (2010)** who showed that the tamarind fruit extract has a hypoglycemic effect in diabetic mice. Also, **Wagh and Bhagure, (2012)** reported that tamarind fruit pulp has antioxidant, anti-inflammatory and antidiabetic effects. **Unnikrishnan *et al.*, (2015)** revealed that tamarind fruit pulp extract, in a dose-dependent manner has restrained effect on both α -glucosidase and α -amylase enzymes. The suppression of α -amylase is an efficacious strategy to reduce postprandial hyperglycemia for diabetes management.

The potential mechanism for the protective effect of AETF against hyperglycemic and testicular damage induced by alloxan could be attributed to its antioxidant protection and lower lipid peroxidation. The antioxidant effect of AETF might be due to its high content of antioxidant polyphenolic compounds as well as catechin, which had been reported to have intense antioxidant activities (**Katalinic *et al.*, 2004**). Phytochemical analysis of tamarind fruit pulp identified the presence of phenolics as naringenin, epicatechin, apigenin, taxifolin, procyanidins, luteolin and eriodictyol (**Sudjaroen *et al.*, 2005**), steroids, saponins, alkaloids, tannins, flavonoids, phenols, glycosides and terpenoids (**Ishaku *et al.*, 2016**). All these compounds were reported to revealed strong antioxidant properties (**Bundy *et al.*, 2008**). Saponins and glycosides were founded to have anti-hyperglycemic and hypoglycemic effects through stimulating insulin release from isolated pancreatic islets (**Grover *et al.*, 2002**) and flavonoids has a strong antioxidant (**Hausteen, 2002**).

Conclusion: AETF produces protective effect and antioxidant activity against alloxan - induced diabetic and reproductive toxicity in

male rats. The protective effect could be attributed to the antioxidant property. Regular intake of tamarind fruit may be beneficial for male patients suffering from diabetic and/or infertility problems and can be suggested as well good for the control of postprandial hyperglycemia.

References

- Aitken, R.J., Roman, S.D. (2008). Antioxidant systems and oxidative stress in the testes. *Oxidative Medicine and Cellular Longevity*. 1(1): 15-24;
- Asmat, U., Abad, K. and Ismail K. (2016). Diabetes mellitus and oxidative stress-A concise review. *Saudi Pharm J.*, 24(5): 547-53.
- Bergmeyer, H.U., Schreiber, P. and Wahlefeld, A.W. (1978). Optimization of methods for aspartate and alanine aminotransferase. *Clin. Chem.*, 24(1): 58-61.
- Bisht, S., Faiq, M., Tolahunase, M. and Dada, R. (2017). Oxidative stress and male infertility. *Nat. Rev. Urol.*, 14(8): 470–485.
- Buko, V., Lukivskayam, O., Nikitin, V., Tarasov, Y., Zavodnik, L., Borodinsky A. and Gorenshtein, B. (1996). Hepatic and pancreatic effects of oleylphosphatidylcholine in rats with alloxan induced diabetes. *Cell Biochem. Funct.* 14(2): 131-137.
- Bundy, R., Walker, A.F., Middleton, R.W., Wallis, C. and Simpson, H. (2008). Artichoke leaf extract (*Cynara scolymus*) reduces plasma cholesterol in otherwise healthy hypercholesterolemic adults: A randomized, double blind placebo controlled trial. *Phytomedicine* 15 (9): 668–675.
- Carleton, H. (1979). *Lodon*, Oxford University Press, New York, USA, Toronto.

- Gaunay, G., Nagler, H.M. and Stember, D.S. (2013). Reproductive sequelae of diabetes in male patients. *Endocrinol Metab Clin North Am.*, 42(4): 899-914.
- Grover, J.K., Yadav, S. and Vats, V. (2002). Medicinal plants of India with anti-diabetic potential. *J Ethnopharmacol.*81(1):81–100.
- Hatanaka, T., Ogawa, D., Tachibana, H., Eguchi, J., Inoue, T., Yamada, H., Takei K, Makino, H., Wada, J. (2016). Inhibition of SGLT2 alleviates diabetic nephropathy by suppressing high glucose induced oxidative stress in type 1 diabetic mice. *J Pharmacol Res Perspect*, 4(4): e00239.
- Hausteen, B. H. (2002). The Biochemistry and medical significance of the flavonoids. *Journal of Pharmacology and therapeutics*, 96(2-3): 67-202.
- Havinga, R.M., Hartl, A., Putscher, J., Prehsler, S., Buchmann, S. and Vogl, C.R. (2010). *Tamarindus indica* L. (Fabaceae): patterns of use in traditional African medicine. *Journal of Ethnopharmacology*, 127(3): 573-588.
- Ishaku, G.A., Ardo, B., Abubakar, H. and Peingurta, F. (2016). Nutritional composition of *tamarindus indica* fruit pulp. *J of Chemistry and Chemical Sciences*, 6(8): 695-699.
- Katalinic´, V., Milos, M., Modun, D., Music´, I. and Boban, M. (2004). Antioxidant effectiveness of selected wines in comparison with (+)-catechin. *Food Chemistry*, 86(4): 593–600.
- Lee, J., Kuchakulla, M., Arora, H., Kulandavelu, S., Gonzalez, E., Masterson, T. Hare, J., Kaiser, U. and Ramasamy, R. (2019). Age Induced nitroso-redox imbalance leads to subclinical hypogonadism in male mice. *Front. Endocrinol.*, 10:1-5.
- Nahar, L., Nasrin, F., Zahan, R., Haque, A., Haque, E. and Mo-saddik, A. (2014). Comparative study of anti-diabetic activity of

- Cajanuscajan* and *Tamarindus indica* in alloxan-induced diabetic mice with a reference to in vitro antioxidant activity. *Pharmacognosy Res*, 6 (2):180-187
- Ohkawa, H., Ohishi, N. and Yagi, K. (1979). Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Analyt. Biochem.*, 95:351–358.
 - Oyedemi, S.O., Bradley, G. and Afolayan, A.L. (2011). Beneficial effect of aqueous stem bark extracts of *Strychnos henninhsii* gilg in streptozotocin-nicotinamide induced type 2 diabetic wister rats. *International Journal of Pharmacology*,7(7): 773-781.
 - Paglia, D.F. and Valentine, W.N. (1976). Studies on glutathione and glutathione characterization of erythrocytes glutathione peroxidase. *J. Lab. Clin. Med.*, 70(1):158-169.
 - Pattar, A., Kukanoor, L., Guranna, P. and Praveen, J. (2013). Standardization of methods for extraction of tamarind pulp. *Karnataka J. Agric. Sci.*,26(4): 570-571.
 - Posario, P. W. (2010). Normal values of serum IGF - I in adults: results from a Brazilian population. *Arq. Bras. Endocrinol. Metabol.*, 45(5):477-481.
 - Prakash, R., Kumar, M., Singh, D.K., Chandra, M. and Verma D. (2010). Anti-diabetic effect of *Morinda citrofolia* and *Coccinia indica* in alloxan induced diabetic rats. *Advances in Bio. Research*.1(1):75-77.
 - Rebar, R.W., Erickson, G.G. and Yen, S.S.C. (1982). Idiopathic premature ovarian failure: clinical and endocrine characteristics. *Fertil. Steril.*, 37(1): 35.
 - Reeves, P., Nielsen, F. and Fahmy, G. (1993). AIN-93. Purified diets for laboratory rodents: Final reports of the American Institute of Nutrition adhoc 9 wriling committee of reformulation of the AIN-76 A Rodent Diet. *J. Nutr.*, 123(11): 1939-1951.
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- Roy, M., Rahman, S., Rehana, F., Munmun, M., Sharmin, N., Hasan, Z., Al Mamun, A., Khatun, A. and Rahmatullah, M. (2010). Evaluation of anti-hyperglycemic potential of methanolic extract of *Tamarindus Indica* L. (Fabaceae) fruits and seeds in glucose-induced hyperglycemic mice. *Advances in Natural and Applied Sciences*, 4(2): 159-162.
- Roy, S.E. (1970). Colorimetric determination of serum alkaline phosphatase. *Clin. Chem.*, 16(5): 431-432.
- Sadoughi, D., Edalatmanesh, M. and Rahbarian, R. (2019). Protective effect of curcumin on quality parameters of sperm and testicular tissue alterations in alloxan-induced diabetic rats as animal model. *The Indonesian Biomedical Journal*, 11(3): 225-337.
- Sedlak, J. and Lindsay, R.H. (1968). Estimation of total, protein-bound, non-protein bound sulfhydryl groups in tissues with Ellman's reagent. *Anal. Biochem.*, 25(1): 192-205.
- Shoorei, H., Khaki, A., Khaki, A., Hemmati, A., Moghimian, M. and Shokoohi M. (2019). The ameliorative effect of carvacrol on oxidative stress and germ cell apoptosis in testicular tissue of adult diabetic rats. *Biomed Pharmacother.* 111: 568-678.
- Sinha, K.A. (1972). Colorimetric assay of catalase enzyme. *Anal. Biochem.*, 47(2): 28- 330.
- Spitz, D.R. and Oberley, L.W. (1989). An assay for superoxide dismutase activity in mammalian tissue homogenates. *Anal. Biochem.*, 179(1): 8-18.
- Sudjaroen, Y., Haubner, R., Wu'ritele, G., Hull, W., Erben G., Spiegelhalder, B., Changbumrung, S., Bartsch, H. and Owen, R. (2005). Isolation and structure elucidation of phenolic antioxidants from tamarind (*Tamarindus indica* L.) seeds and pericarp. *Food and Chemical Toxicology*, 43(11): 1673–1682.

- Tietz, N.W. (1995). Clinical Guide to Laboratory Tests, 3rd Edition, W.B. Saunders, Co., Philadelphia. 268-580
- Unnikrishnan, P.S., Suthindhiran, K. and Jayasri, M. (2015). Alpha-amylase inhibition and antioxidant activity of marine green algae and its possible role in diabetes management. Pharmacogn Mag.11(Suppl 4):511–515.
- Vijay, S., Ghongane, B., Nayak, B. Raul, A., Kumar, A., Mutalik, M. and Kapure, N. (2012). An experimental study on effect of antioxidant vitamin E in stress and alcohol induced changes in male fertility in albino rats. Int J Med Res Health Sci.,1(1):1-6.
- Wagh, A. and Bhagure, L. (2012). A Pharmacological review on *Tamarindus indica* Linn (*Caesalpinaceae*). Int. J. of Universal Pharmacy and Life Sciences, 2(1):60-64.

**الأدوار الواقية المحتملة للمستخلص المائي للّب فاكهة التمر هندي ضد ارتفاع سكر
الدم وسمية الخصية في ذكور الجرذان المصابة بداء السكري / المُحدث بالالوكسان
امل فوزي محمود الجزار
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المُلخّص

أجريت الدراسة الحالية بهدف تقييم التأثيرات الواقية المُحتملة للمستخلص المائي للّب ثمار التمر الهندي ضد ارتفاع مستوي السكر في الدم ومشاكل الخصوبة في ذكور الجرذان المصابة بداء السكري. تم الحصول على المستخلص المائي من لب ثمار فاكهة التمر الهندي عن طريق نقعه في ماء مقطر بنسبة ١ : ٢ (لب الفاكهة: ماء) لمدة ١٢ ساعة . بعد فترة النقع تم تسخين المحلول لمدة ٢٠ دقيقة عند ٥٠ درجة مئوية، ثم ترشيح المحلول وتركيزه تحت ضغط منخفض. في التجربة الحالية، تم استخدام خمسة وثلاثين جرذ ذكر ابيض وتم تقسيمها إلى خمس مجموعات (كل مجموعة = ٧ جرذان). استخدمت المجموعة (١) كجرذان طبيعية ، بينما استخدمت المجموعة (٢) كجرذان مصابة بداء السكري وغير معالجة والمجموعات (٣) و (٤) و (٥) المصابة بالسكري تم إعطاؤهم المستخلص المائي لفاكهة التمر هندي عن طريق الفم بجرعة ٢،١ و ٣ مل / ١٠٠ جم من وزن الجسم / يوم ، على التوالي. أظهرت النتائج ان تناول المستخلص المائي لفاكهة التمر هندي عن طريق الفم للجرذان المصابة بداء السكري إلى تحسن معنوي في وزن الخصيتين والحويضلات المنوية و غدة البروستاتا كمقارنة بالجرذان المصابة بالسكر والغير معالجة. كما أدى إلى انخفاض معنوي في مستوي سكر الدم، وأنشطة إنزيمات أسبارتات أمينوترانسفيراز و أللأنين أمينوترانسفيراز والاكلينفوسفاتياز وزيادة في مستويات الأنسولين والتستوستيرون و LH و FSH في سيرم الدم ، كمقارنة بالفئران المصابة بالسكر والغير معالجة. كما أدى المستخلص المائي لفاكهة التمر هندي إلى تحسن ملحوظ في مستوي المالونداي الدهيد ونشاط إنزيمات الجلوتاثون ،الجلوتاثون بيرواوكسيديز،السوبراوكسيد ديسميوتاز و الكتاليز في أنسجة الخصيتين للجرذان المصابة بداء السكري. و أظهر الفحص التشريحي للخصيتين في الجرذان المصابة بمرض السكري والمعالجة بنية نسجية شبه طبيعية للأنابيب المنوية مع تجويف مليء بالحيوانات المنوية، خاصة مع الجرعات العالية من المستخلص المائي لفاكهة التمر هندي. في الخلاصة: أظهر المستخلص المائي لفاكهة التمر هندي نشاطاً خافضاً لسكر الدم ومضاداً للأكسدة وتأثيراً وقائياً ضد مشاكل العقم في ذكور الجرذان المصابة بداء السكري.

الكلمات المفتاحية: فاكهة التمر الهندي، الإنزيمات المضادة للأكسدة، الهرمونات الجنسية، داء السكري، الفحص الهستوباثولوجي