Egypt. J. Vet. Sci. **Vol. 56, (Special issue),** pp. 847-859 (2025)

Egyptian Journal of Veterinary Sciences

https://ejvs.journals.ekb.eg/

Cape Gooseberry (*Physalis peruviana* L.) Fruit Juice Defeats Testicular Impairment in Type I Diabetic Rats: Targeting Inhibition of Advanced Glycation End Products, Oxidative Stress, and Inflammation

Safia M. A. Bahshwan[1], Haya M. Aljadani[2], Afnan H. Saaty[2], Eman A. Abduljawad[2], Manal M.S. Mansoury[2*], Saleh A. Alharbi[3], Ahlam A. Alahmadi[4] and Soad Shaker Ali[5]

[1] *Biological Sciences Department, College of Science and Arts, King Abdulaziz University (KAU), Rabigh 21911, Saudi Arabia (SA).*
[2] *Department of Food and Nutrition, Faculty of Human Sciences and Design, KAU, Jeddah, SA.*
[3] *Laboratory Department, Eradah Services, Second Health Cluster, Eradah & Mental Health Complex, Ministry of Health, Jeddah, SA.*
[4] *Department of Biological Sciences, Faculty of Science, KAU, Jeddah, SA.*
[5] *Department of Histology and cell Biology, Faculty of Medicine, Assiut University, Member as Researcher in Yousef Abdul Latiff Jameel.*

Abstract

METABOLIC diseases, as diabetes type 1 (DT1), frequently correlate with diminished male fertility and testicular damage. The primary pathogenic mechanisms were elevated blood glucose levels, disrupting the hypothalamus–pituitary–testis axis, mitochondrial dysfunction, chronic inflammation, oxidative stress, and the build-up of advanced glycation end products (AGEs). Documented influence of oxidative stress on fertility has shed light on the use of antioxidant compounds to guard against DT1-induced testicular damage. Cape gooseberry fruit juice (CGFJ) boosts the antioxidant defence systems against reproductive toxicity. This study aimed to assess the probable preventive impact of CGFJ against testicular injury related to DT1 in rats. Furthermore, evaluate the impact of CGFJ on AGEs and indicators of oxidative stress. Male rats (five groups) were randomly allocated as follows: 1- control; 2- STZ-diabetic; 3- MT-treated diabetic; 4- CGFJ-treated diabetic; 5- CGFJ + MT-treated diabetic groups. DT1 was induced with a single intraperitoneal injection of STZ (65 mg/kg). Metformin (MT) (600 mg/kg) and CGFJ (5mL/kg) were orally administered once/day for 70 days. The findings demonstrated that GBFJ reduced serum glucose and elevated serum insulin and testosterone levels, and ameliorated serum gonadotrophic hormones. GBFJ administration alleviated the pathological features of testicular injury made by STZ. The GBFJ decreased oxidative stress levels, as evidenced by reduced MDA levels, and improved antioxidant enzyme activity (CAT and GR). Additionally, GBFJ improved serum levels of AGEs and the testicular protein expression of NF-κB. GBFJ significantly augments the effects of MT, it mitigated DT1-induced testicular damage *via* inhibiting AGEs, inflammation, and oxidative stress.

Keywords: AGEs, Cape gooseberry, Diabetes type 1, Inflammation, Oxidative stress.

Introduction

The figure for worldwide diabetes type 1 (DT1) is increasing rapidly. In 2025, there are an estimated 9.5 million subjects diagnosed with DT1 all over the world, in contrast to 8.4 million subjects in 2021, with a 13% rise [1]. DT1 is a chronic cumulative autoimmune illness distinguished by drastic injury to pancreatic β cells, producing a significant decline of insulin and subsequent high blood glucose levels [2,3]. Persistent hyperglycemia may result in numerous problems, such as nephropathy [4], neuropathy [5], cardiomyopathy [6], and sexual dysfunction [7]. *In vivo* and *in vitro* research revealed that insulin possess a beneficial impact on the spermatogenesis and gonadal tasks [8,9]. Insulin insufficiency, the lineament of DT1, might thus seriously affect male sexual function [10]. The mechanisms through which DT1 influences male reproductive function have not been entirely understood, but the hypotheses stem from the outcomes of raised blood glucose level (BGL) and reduced insulin secretion on spermatogenesis, the hypothalamic/testicular axis, and the seminal content of reactive oxygen species (ROS) [11].

*Corresponding authors: Manal M.S. Mansoury, E-mail: mmmansouri@kau.edu.sa, Tel.: 00966505697927
(Received 06 October 2025, accepted 23 October 2025)
DOI: 10.21608/ejvs.2025.429004.3169

Advanced glycation end products (AGEs) are a set of various compounds that are produced upon glucose reduction and are incorporated with proteins, lipids, or nucleic acids in the lack of enzymatic assistance. They are connected to one or more of their numerous receptors (RAGE) found on various types of cells; leading to a pathway of harmful biological responses to human health [12]. The increase of AGEs is a vital factor that assists in the aggravation of complications related to DT1. These AGEs worsen inflammation and oxidative stress by engaging with the RAGE. AGEs are involved in the pathogenesis of microvascular and macrovascular issues in DT1. Prolonged inflammation and oxidative stress boost resistance to insulin and hinder diabetes management [13]. The key pathogenic factors under diabetes-associated testicular harm include raised BGL, ROS, chronic inflammation, mitochondrial dysfunction, and the build-up of AGEs [14].

With the evolution of investigation on AGEs, the impact of AGEs on reproductive systems has gained increasing interest in latest years [15]. The pathogenesis of men's infertility is complex. Reduced sperm quality, testicular dysfunction, impotency, and abnormal endocrine factors such as deficiency in LH or FSH all denote male infertility. Reduced sperm number and viability are well-known features of male abnormal fertility. Recently, AGEs have been recognized to complicate male fertility, with oxidative stress being among the crucial causes [16].

Oxidative stress is among the principal precursors of infertility, regardless of its origin [17], as it brings germ cells and testicular somatic to trigger overproduction of ROS, exceeding the body's basic defence against ROS and breeding destruction to numerous cellular macromolecules and structures [18]. In this view, spermatozoa are specifically vulnerable to oxidative injury owing to the great content of polyunsaturated fatty acids (PUFA) in their membrane of the cell and the reduced amount of antioxidants in their cytoplasm, causing a marked lessening in sperm number and semen character [18]. The vital influence of oxidative stress on fertility is also emphasized by the evidence that plenty of treatment approaches to guard against infertility are mainly directed to decrease oxidative stress—for instance, utilizing antioxidant compounds [19].

Physalis peruviana L., a member of the Solanaceae family, commonly named Cape gooseberry is an exotic small orange berry fruit safeguarded by a calyx (a papery husk). CG taste is sour and sweet. As it matures, its acidity increases with a decrease in pH [20]. CS is highly intriguing as a promising fruit due to its exceptional nutritional value and abundant bioactive compounds [21]. Its fruit contains many therapeutic phytochemical polyphenolic components such as alkaloids, tannins, phenolic acids, flavonoids, physalins, carotenoids, withanolides, phenolics, and phytosterols, as well as vitamins C, which contribute to its physiological and therapeutic effects [22]. Cape gooseberry has antioxidant, antiulcer, anti-inflammatory, antitumor, hepatoprotective, and antihyperlipidemic properties [23]. A study of Abdel Moneim [24] reported that Cape gooseberry juice enhances the antioxidant defence mechanism against reproductive toxicity induced by CCl4 and could have a treating effect in free radical-mediated diseases and infertility. Nowadays, there is an increased focus on Cape gooseberry fruit as a herbal source of antioxidants, in an attempt to preserve the human body from oxidative stress and impede the progression of several long-standing disorders [25]. Furthermore, Cape gooseberry peels and fruit enhance the glucose homeostasis, increase insulin secretion, improve hyperlipidemia, as well as prevent diabetes-associated hepatotoxicity [26].

This study focused on the probable protective impact of Cape gooseberry fruit juice (CGFJ) against testicular damage associated with DT1 in rats. Additionally, the study will assess how CGFJ affects AGEs and markers of oxidative stress.

Material and Methods

Preparation of Cape gooseberry fruit and peel juice

Cape gooseberry ripe fruits were purchased from Kom Hamada Farm, Al-Buhayrah, Egypt, from February to April 2024. Cape gooseberry was validated for identification via herbarium specimens at the herbarium of the Botany Department, Agricultural Research Centre (ARC), Giza, Egypt. Cape gooseberry ripe intact fruits with brilliant orange color were chosen. Whole Cape gooseberry fruits were washed and dried. The fruits' pulp was mixed for 5 min by the blender (Moulinex, France), and then the mixture was filtered through cheesecloth [27]. The Cape gooseberry fruit juice (CGFJ) was freshly prepared for use.

Gas chromatography–mass spectrometry (GC–MS) analysis

Active constituents of Cape gooseberry fruit were analysed using GC-MS. The database was used to interpret and identify the phytoconstituents of Cape gooseberry fruit by comparing the mass spectrum of unknown constituents with the spectrum of the known constituents stored in the library database of the instrument.

Induction of DT1

As stated, in Zeng et al. [2021], rat model of DT1 was constructed [28]. Diabetic rats were prepared by fasting them overnight, then giving an intraperitoneal (I.P.) injection of 65 mg/kg STZ (STZ from Sigma-Aldrich, St. Louis, MO, USA) dissolved in a citrate buffer (0.1 mol/L, pH 4.5) the day of the experiment. To prevent the rats from dying from the STZ injection, and hypoglycemia related to fasting, 5%

glucose solution was provided. Diabetic rats were prepared by following up with rats that had a fasting BGL with ≥ 250 mg/dl after 72 h then, were used for the remaining experiments [29].

Experimental design

The experimental protocol in this research was carried out accordance with the guidelines for the use and care of laboratory animals [30]. A total of fifty-eight male (200-250 g) Wistar rats were obtained and housed in standard lab conditions in the Animal Unit, ARC, Egypt. Rats were fed on a standard diet and allowed to have free water. After the adaptation period (one week), rats were divided randomly into five groups. The first group was the control (n=10) (non-treated and non-diabetic rats) (rats were I.P. injected with PBS); the second was the STZ-diabetic rats (n=14); the third was MT-treated diabetic rats (n=10), metformin (MT) was dissolved in distilled water and given orally at a dose of 600 mg/kg [31], (MT from Sigma-Aldrich, St. Louis, MO, USA). The fourth group was CGFJ-treated diabetic rats (5 ml/kg/ oral dose) (n=12) [31]. The fifth was CGFJ + MT treated diabetic rats (n=12).

On day 70, samples of blood were collected from eight rats from each group. The samples were kept to coagulate, centrifuged at 4000 rpm for 10 min, and then serum samples were kept at -80 °C for estimation of glucose, hormonal assay, advanced AGEs, and redox status. Testis tissue samples were preserved in formalin (10%) for histopathological and immunohistochemical examinations.

Estimation of serum glucose and insulin

Glucose concentration was estimated by colorimetric kits (BioMérieux, France). The insulin hormone was estimated by an ELISA kit (My BioSource, San Diego, CA 92185-3308, USA).

Estimation of serum testicular hormones

Concentrations of testosterone (TST), follicle-stimulating hormone (FSH), and luteinizing hormone (LH) were assessed using ELISA kits (LDN Labor Diagnostika Nord GmbH Com Germany, Abnova, Taiwan, and Kamiya BioMed Com USA, respectively) as in the kits' procedures.

Histopathology examination

Testis specimens, after fixation in formalin, were prepared following the standard technique, and then stained with hematoxylin and eosin (H & E stain) for examination under a high-performance light microscope (x 200 and x 50 magnification).

Quantitative scoring of testis lesions

The score of testicular lesions was estimated at x 200 magnification according to Johnsen's score. The histopathological scoring of the testicular lesions was categorised on a 10-point rating according to the absence or existence of the primary cell types arranged on the base of maturity. 10, 9, or 8 scores demonstrate the presence of spermatozoa; 7 or 6 scores demonstrate the presence of spermatids (and no further); 5 or 4 scores demonstrate the presence of spermatocytes (and no further); 3 only spermatogonia, 2 only Sertoli cells, and 1 no cells [33].

Immunohistochemical examination

The immunoreactivity of nuclear factor kappa beta (NF-κB) was evaluated semi-quantitatively by performing the immunohistochemistry procedure [34]. Briefly, NF-κB/p65 antibody was applied to sections with the appropriate dilutions specified in the guidelines. After the incubation period with anti-NF-κB/p65 monoclonal antibody (Santa Cruz Biotechnology, USA). The slides were rinsed 3 times with PBS, then were treated with a goat anti-rabbit secondary antibody (Cat No. K4003, EnVision +TM System Horseradish Peroxidase Labelled Polymer, Dako, USA). Then the slides were counterstained with hematoxylin and dried for examination.

Estimation of AGEs

Serum AGEs concentration was determined using the Sandwich ELISA kit LSBio (LifeSpan Biosciences, USA) as in the kits' procedure.

Estimation of the redox status

Serum glutathione reductase (GR), catalase (CAT), and malondialdehyde (MDA) were assessed through ELISA kits LSBio (LifeSpan Biosciences, USA) as described in the kits' procedure.

Statistical tests

Results were displayed as mean ± SD. All statistical analysis was done using one-way analysis of variance (ANOVA), followed by Tukey's test. The GraphPad program Prism 8 software was used in performing the statistical tests. P-value ≤ 0.05 was reflected significant.

Results

The GC–MS analysis

The chromatogram of the CG-MS`s phytoconstituents is presented in Fig. 1, while the interpretation and identification of these phytoconstituents with their retention time (RT) and peak area (%) are presented in Table 1. Nineteen peaks in the chromatogram were identified in CG-MS during the 22.56 min measurement period. The major constituents were 3-O-methylgallic acid (14.54), phloroglucinol (13.25), and 2-hexadecanol (12.99) separated at the RTs 8.594, 12.994, and 11.415 min, respectively. Followed by phytol (7.76%), 3',4',7-trimethylquercetin (6.87%), nonacosanol (6.64), vitexin (6.23%), and phytanic acid (5.44%) separated at the RTs 15.155, 15.692, 22.233, 14.766, and 19.924 min, respectively.

Serum glucose and insulin concentrations

There was a substantial rise in glucose with an extensive decline in insulin in STZ-diabetic rats relative to the control rats ($p \leq 0.001$). STZ-diabetic rats received CGFJ or MT had substantial decrease in glucose with substantial elevation in insulin levels relative to the STZ-diabetic group ($p \leq 0.001$). There was a substantial difference between STZ-diabetic group received CGFJ and STZ-diabetic group received MT ($p \leq 0.05$). However, STZ-diabetic rats co-received CGFJ and MT had a significant noticeable amelioration in glucose and insulin levels relative to STZ-diabetic rats ($p \leq 0.001$), as well as with the STZ-diabetic group that received CGFJ ($p \leq 0.001$) and the STZ-diabetic group that received MT ($p \leq 0.05$). Interestingly, there were insignificant changes between STZ-diabetic rats co-received CGFJ and MT and the control group (Fig. 2).

Serum sex hormones

As demonstrated in Table 2, the STZ-diabetic rats showed a substantial decline in TST hormone along with substantial rise in LH and FSH hormone levels relative to the control group ($p \leq 0.001$). The STZ-diabetic group received CGFJ or MT, had a significant elevation in TST hormone, along with a significant decline in FSH and LH hormone levels relative to the STZ-diabetic group ($p \leq 0.001$). However, the more pronounced effect was detected in the STZ-diabetic rats that co-received CGFJ and MT; they had insignificant changes relative to the control group. Additionally, their values were significantly different relative to STZ-diabetic rats ($p \leq 0.001$), as well as with the STZ-diabetic group that received CGFJ ($p \leq 0.05$) and the STZ-diabetic group that received MT ($p \leq 0.01$).

Testicular histopathology examination and quantitative scoring of its lesions

The testis sections for control rats showed normal seminiferous tubules and various layers of spermatogenic cells with free sperms within seminiferous tubules (Fig. 3A & 4A). In the STZ-diabetic group, testis sections revealed severe damage of the seminiferous tubules, degenerative changes, and atrophy of spermatogenic cells, most of which had pycnotic nuclei, as well as interstitial oedema (Fig. 3B & 4B). In the MT-treated STZ diabetic group, the sections showed a notable improvement of degenerative changes in the spermatogenic cells and an increase in the number of sperms in the lumen, and at high power a normal seminiferous tubules were observed (Fig. 3C & 4C). In the STZ diabetic group treated with CGFJ, there was an improvement of degenerative changes in the spermatogenic cells and an increase in spermatogenesis as well, and at high power a normal seminiferous tubules were observed (Fig. 3D & 4D). In the group STZ diabetic treated with CGFJ+MT, testis sections showed a remarkable improvement of degenerative changes in the spermatogenic cells and an obvious increase in the number of sperms in the lumen, almost like the control group (Fig. 3E & 4E).

Histological examination was used to quantitatively score testicular lesions according to the scoring system that represents the presence or absence of the main cell types arranged in the order of maturity. The control group had the highest score (10), which demonstrates the presence of spermatozoa. The STZ-diabetic group had significantly lower scores (3.88) relative to the control group, which demonstrates the presence of spermatogonia and Sertoli cells. The STZ-diabetic groups received CGFJ, or MT had significant increase in the scores (6.63 and 6.75, respectively) relative to the STZ-diabetic group ($p \leq 0.001$), which demonstrates the presence of spermatids. However, STZ-diabetic rats co-received CGFJ and MT had noticeable improvement in the scores (9.36), with significant differences relative to the STZ-diabetic group, as well as STZ-diabetic rats that received CGFJ or MT ($p \leq 0.001$). Co-received CGFJ and MT successfully augmented the testis structure, leading to normalization of testicular histopathological lesions, with insignificant change relative to the control rats (Fig. 5).

Testicular immunohistochemical examination of NF-κB

In the control sections, a negative reaction of NF-κB in spermatogenic cells and interstitial Leydig cells was observed (Fig. 6A & 7A). In the STZ-diabetic group sections, a severe expression of NF-κB with strong positive reaction in spermatogenic cells and interstitial Leydig cells (Fig. 6B & 7B). In MT-treated STZ diabetic sections, mild expression of NF-κB with weak positive reaction in some spermatogenic cells (Fig. 6C & 7C). In CGFJ-treated STZ diabetic sections, moderate expression of NF-κB with positive reaction in some of the spermatogenic cells (Fig. 6D & 7D). In CGFJ+MT-treated STZ diabetic sections marked decline in expression of NF-κB with negative reaction in spermatogenic cells, similar to the control group (Fig. 6E & 7E).

Serum AGEs

There was a substantial increase in serum AGEs levels in STZ-diabetic group relative to the control group. The STZ-diabetic group that received CGFJ or MT had substantial decline in AGEs levels relative to the STZ-diabetic group ($p \leq 0.001$). There was no substantial difference between the STZ-diabetic group that received CGFJ and the STZ-diabetic group that received MT. However, STZ-diabetic rats co-received CGFJ and MT had a substantial noticeable decrease in AGEs levels compared to the STZ-diabetic rats ($p \leq 0.001$), as well as with the STZ-diabetic group that received CGFJ ($p \leq 0.05$) and the STZ-diabetic group that

received MT ($p \leq 0.01$). Additionally, there were non-significant changes between the STZ-diabetic rats that co-received CGFJ and MT and the control group (Fig. 8).

Serum redox status

As demonstrated in Table 3, the STZ-diabetic rats exhibited a substantial oxidative stress in rats, there were a substantial decline in CAT and GR activities along with a substantial rise in MDA level relative to the control group ($p \leq 0.01$). STZ-diabetic group that received CGFJ or MT had a substantial elevation in CAT and GR activities along with a substantial decrease in MDA levels compared to the STZ-diabetic group ($p \leq 0.001$). However, the more pronounced effect was detected in the STZ-diabetic rats that co-received CGFJ and MT; they had non-significant changes relative to the control group. Additionally, their values were significantly different compared to the STZ-diabetic rats ($p \leq 0.001$), as well as with the STZ-diabetic group that received CGFJ ($p \leq 0.05$) and the STZ-diabetic group that received MT ($p \leq 0.01$).

Discussion

In the current investigation, STZ-induced DT1 resulted in significant testicular injury, evidenced by pronounced degenerative alterations and atrophy of the spermatogenic cells, damage to the seminiferous tubules, alongside a significantly diminished Johnsen's score, indicating the exclusive presence of spermatogonia and Sertoli cells. Consistent with our findings, recent studies have confirmed that the STZ-induced diabetes model results in testicular degeneration, characterized by damage to the seminiferous tubules and a reduction in spermatogonia cells. Furthermore, they reported an extensive decline in the diameter of the seminiferous tubules, accompanied by structural deterioration and a marked decline in the average Johnsen's score [35-37].

It is generally known that DT1 causes an excess of oxidative stress because high BGL triggers exceptional mechanisms that make ROS levels rise in numerous tissues in humans, including the testis [19]. In this study, we validated the induction of DT1-caused oxidative stress, as implied by raised MDA levels, an indicator of lipid peroxidation (LPO), and the diminished function of the antioxidant enzymes CAT and GR. Additionally, another study showed that a diabetic model caused by STZ elevated oxidative stress, which led to testicular degeneration [35]. Oxidative overload is a contributing factor for spermatogenesis harm in DT1, as elevated glucose levels produce high levels of ROS that severely compromise the sperm membrane, resulting in LPO and the generation of MDA [38].

DT1 is marked by a close link between inflammation and oxidative stress, since oxidative stress activates inflammatory processes. Moreover, certain investigations have demonstrated that oxidative stress and inflammatory responses induced by diabetes in testicles tissue [39,40]. We have shown that DT1 increased the expression of NF-kB in the testicles, which confirms that the inflammatory pathway was activated. It is widely known that NF-kB is the main player in activating inflammation, as the majority of genes that code for proinflammatory cytokines include NF-kB-binding sites in their promoter/enhancer domains [41].

The present findings indicate that STZ-induced DT1 elevated serum AGEs. Higher blood contents of AGEs have been shown as predictors of all complications in individuals with DT1 [42]. AGEs have been demonstrated to induce inflammatory reactions and oxidative stress via their interaction with their receptor, RAGE. Inflammation and oxidative stress have been demonstrated to hinder spermatogenesis and harm spermatozoa; hence, AGEs may likely be a contributing factor in sperm dysfunction in diabetes [42, 44]. The formation of AGEs in the epididymis can induce oxidative stress through receptor interactions, resulting in the creation of ROS via the activation of NADPH oxidase, thereby exacerbating DNA damage and undermining sperm health [45]. AGE buildup leads to diabetes complications by causing direct destruction of tissues and activating RAGE. The interaction between AGE and RAGE leads to an increase in the creation of ROS and the activation of NF-κB, which encourages the secretion of numerous inflammatory factors associated with diabetic problems [42].

The results demonstrated that GBFJ lowered BGL, raised serum insulin and testosterone levels, and improved serum gonadotrophic hormones (LH and FSH). GBFJ treatment lessened the deleterious effects of STZ on the testes. The findings indicated that GBFJ diminished oxidative stress levels, as demonstrated by decreased MDA levels and enhanced antioxidant enzyme activity (CAT and GR). Also, GBFJ raised the concentrations of AGEs in the blood and the quantity of NF-κB in testicles. In our results side, the intake of GBFJ enhanced testicular antioxidant status and reduced lipid peroxidation in CCl_4 -induced reproductive injury. It preserved germ and Leydig cells, promoted spermatogenesis, and provided notable protection against CCl_4 -related fertility impairment [24]. Furthermore, similarly, in cadmium-exposed rats, cap gooseberry extract reduced oxidative stress markers, enhanced antioxidant enzymes and testosterone, and protected testicular tissue by preventing germ cell apoptosis and structural damage [46].

Research on animals indicates that Cap gooseberry may possess possible anti-diabetic properties [47]. A study on animals found that Cape

gooseberry juice decreased the blood sugar level and made insulin resistance better in rats with DT2. Additionally, a meta-analysis revealed that gooseberry supplementation exhibited certain positive effects on blood glucose levels in diabetic individuals [48, 49]. Another study done in India found that those who took 1–3 g of gooseberry powder every day for 21 days had far lower fasting and post-meal blood sugar readings than people who didn't take the powder [50].

GBFJ protects the testicles mainly by eliminating ROS, which boosts antioxidant defences and lowers injury caused by the effects of oxidative stress. It has more ascorbic acid (46 mg/100 g) than typical fruit juices, including peach, apple, and pear [51]. GBFJ has a lot of phenolic components, like kaempferol, quercetin, and myricetin, which are all recognized antioxidants that aid in reducing oxidative damage and potentially prevent problems from diabetes [52-53]. Treatment with kaempferol dramatically decreased oxidative stress and hyperglycemia by inhibiting the AGE-RAGE axis signaling [54]. Kaempferol might halt the development of AGEs by trapping methylglyoxal, which is a toxic intermediate [55]. Kaempferol inhibits NF-κB activity [56].

Conclusion

The present investigation indicated that CGFJ may safeguard against diabetic testicular injury by diminishing oxidative stress, inflammation, and AGE. Furthermore, the combination of CGFJ with the widely used diabetic medicine MT seemed to enhance its protective impact against testicular damage. These results show that CGFJ might be a valuable natural remedy for treating DT1 and associated complications, together with standard medicines. Nonetheless, additional clinical trials are required to validate these encouraging findings.

Funding statement

This study didn't receive any funding support

Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

Ethical of approval

This study follows the ethics guidelines of the Regional Centre for Food and Feed, Agricultural Research Centre, Giza, Egypt (ethics approval number; 21529).

TABLE 1. Phytoconstituents of GBFJ using GC-MS.

No	Phytconstituents	RT (min)	Peak area (%)
1	1-Pyrroline, 2-phenyl	7.52	4.18
2	3-O-Methylgallic acid	8.594	14.54
3	2-Hexadecanol	11.415	12.99
4	Phloroglucinol	12.994	13.25
5	Hexa-hydro-farnesol	14.228	4.17
6	Vitexin	14.766	6.23
7	Phytol	15.155	7.76
8	3',4',7-Trimethylquercetin	15.692	6.87
9	5,7,3',4'-Tetramethoxyflavone	16.644	0.81
10	β-Citronellol	17.288	2.39
11	Geranyl isovalerate	17.57	1.32
12	5,7,3',4'-Tetramethoxyflavone	18.493	0.74
13	Heptacosane	18.838	1.15
14	Phytanic acid	19.924	5.44
15	Salicylic acid β-D-O-glucuronide	20.31	3.47
16	6-C-β-D-Glucosyl-8-C-α-L-arabinosylapigenin	21.413	1.55
17	Quercetin 4'-O-β-D-glucopyranoside	21.61	3.89
18	Nonacosanol	22.233	6.64
19	3,2',4',5'-Tetramethoxyflavone	22.561	2.6
Non-detected phytoconstituents		>22. 56	0.01

Retention time (RT)

TABLE 2. Effect of CGFJ, MT, and CGFJ+MT on serum sex hormon levels in diabetic rats

Groups	**TST** (ng/mL)	**FSH** (ng/mL)	**LH** (ng/mL)
Control	4.77 ± 0.36	21.54 ± 2.69	9.86 ±0.83
STZ-diabetic	2.67 ± 0.40 $^{a\#}$	38.31± 3.11$^{a\#}$	14.82 ± 1.27$^{a\#}$
MT-treated diabetic	3.70 ± 0.41 $^{a\#, b\#}$	29.78 ± 3.95$^{a\#, b\#}$	12.25 ± 1.17$^{a\#, b\#}$
CGFJ- treated diabetic	3.97 ± 0.50 $^{a\#, b\#}$	28.66 ± 4.81$^{a\#, b\#}$	11.84 ± 1.14$^{a\#, b\#}$
CGFJ+ MT-treated diabetic	4.49 ± 0.57 $^{b\#, c*, d\wedge}$	24.86 ± 2.55 $^{b\#, c*, d\wedge}$	10.41 ± 1.11 $^{b\#, c*, d\wedge}$

Data were tabulated as mean ± SD (8/each group). [a]Significant relative to control. [b]Significant relative to STZ-diabetic. [c]Significant relative to MT- treated diabetic. [d]Significant relative to CGFJ-treated diabetic. ($^{\wedge}p \leq 0.05$, * $p \leq 0.01$, and $^{\#}p \leq 0.001$).

TABLE 3. Effect of CGFJ, MT, and CGFJ+MT on serum redox status in diabetic rats

Groups	GR (U/L)	CAT (U/L)	MDA (nmol/mL)
Control	70.38 ± 7.91	7.08 ± 0.57	96.64 ± 11.73
STZ-diabetic	33.54 ± 6.92 $^{a\#}$	3.03 ± 0.47 $^{a\#}$	387.21 ± 33.06 $^{a\#}$
MT-treated diabetic	57.63 ± 3.02 $^{a\#, b\#}$	5.37 ± 0.89 $^{a\#, b\#}$	158.80 ± 14.25 $^{a\#, b\#}$
CGFJ- treated diabetic	61.38 ± 3.54$^{a\#, b\#}$	5.83 ± 0.66 $^{a\#, b\#}$	141.53 ± 16.74 $^{a\#, b\#}$
CGFJ+ MT-treated diabetic	68.00 ± 5.73$^{b\#, c*, d\wedge}$	6.62 ± 0.67 $^{b\#, c*, d\wedge}$	115.75 ± 10.70 $^{b\#, c*, d\wedge}$

Data tabulated as mean ± SD (8/each group). [a]Significant relative to control. [b]Significant relative to STZ-diabetic. [c] significant relative to MT-treated diabetic. [d] Significant relative to CGFJ-treated diabetic. (* p ≤ 0. 01, $^{\wedge}$ p ≤ 0.01, and $^{\#}$p ≤ 0.001).

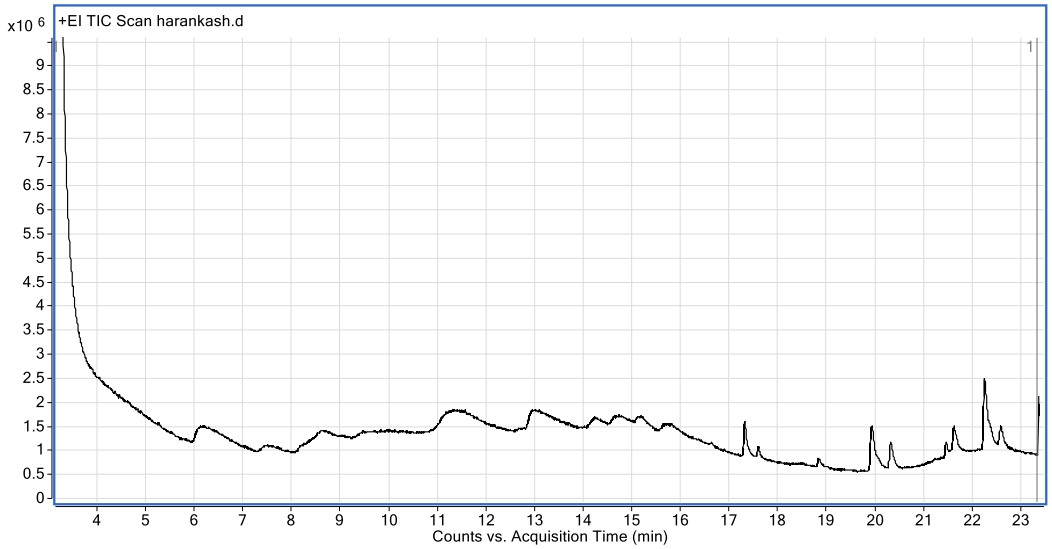

Fig 1. Chromatogram of phytoconstituents of GBFJ using GC-MS

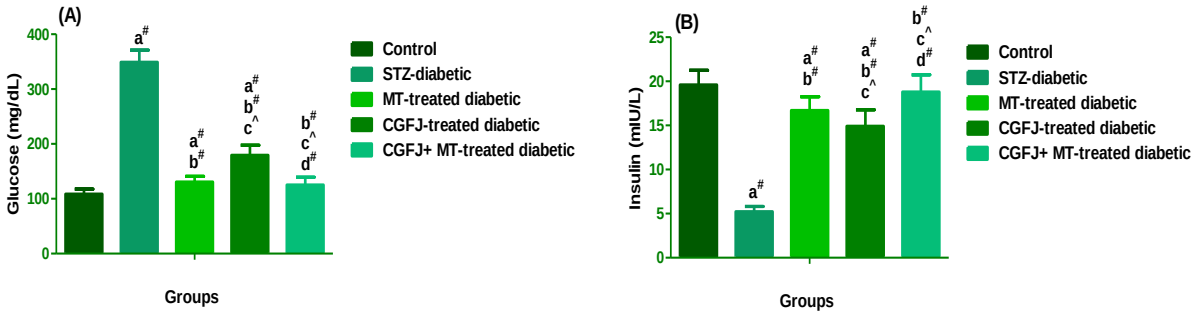

Fig. 2. Effect of CGFJ, MT, and CGFJ+MT on glucose and insulin levels in diabetic rats

Data were presented as mean ± SD (8/each group). [a]Significant relative to control. [b]Significant relative to STZ-diabetic. [c]Significant relative to MT-treated diabetic. [d]Significant relative to the CGFJ-treated diabetic. ($^{\wedge}$ p ≤ 0.05 and $^{\#}$ p ≤ 0.001).

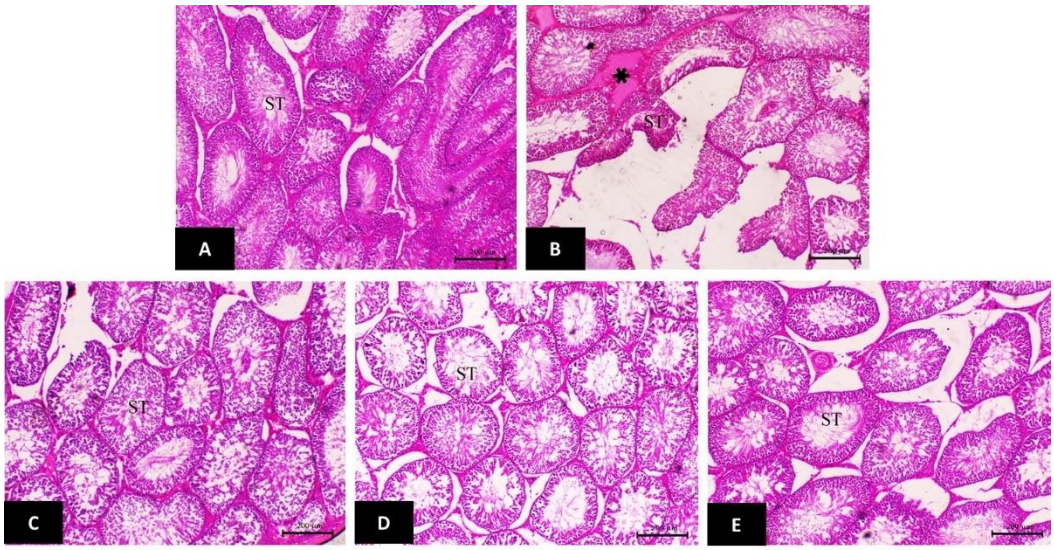

Fig. 3. Photomicrograph of testis sections (H&E X 50, bar=200 µm). (A) Control group; (B) STZ-diabetic untreated group; (C) MT-treated STZ diabetic group; (D) CGFJ-treated STZ diabetic group; (E) CGFJ+MT-treated STZ diabetic group.

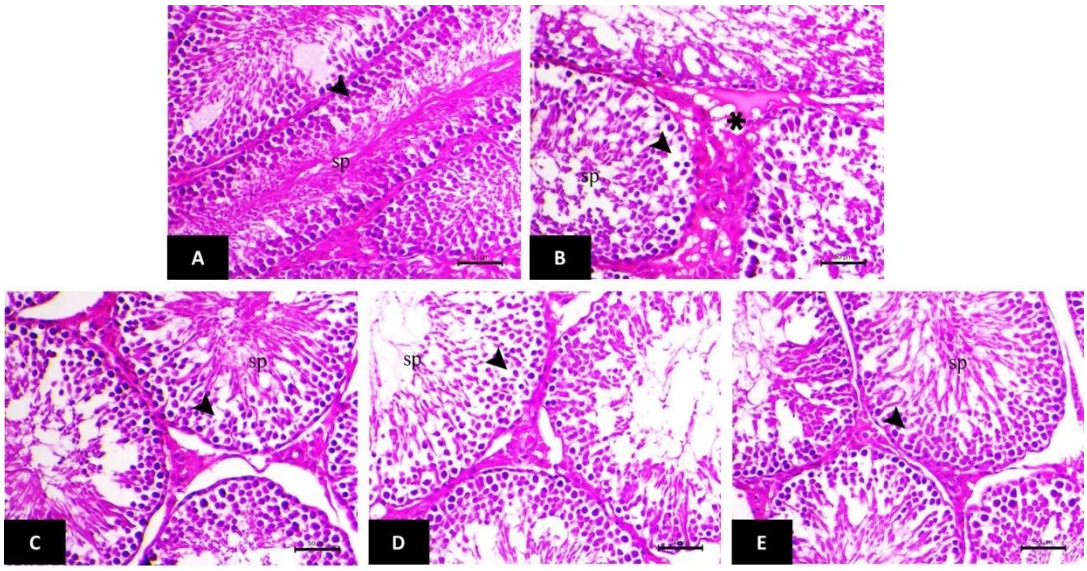

Fig. 4. Photomicrograph of testis sections (H&E X 200, bar=50 µm). (A) Control group; (B) STZ-diabetic untreated group; (C) MT-treated STZ diabetic group; (D) CGFJ-treated STZ diabetic group; (E) CGFJ+MT-treated STZ diabetic group. ST: Seminiferous tubules

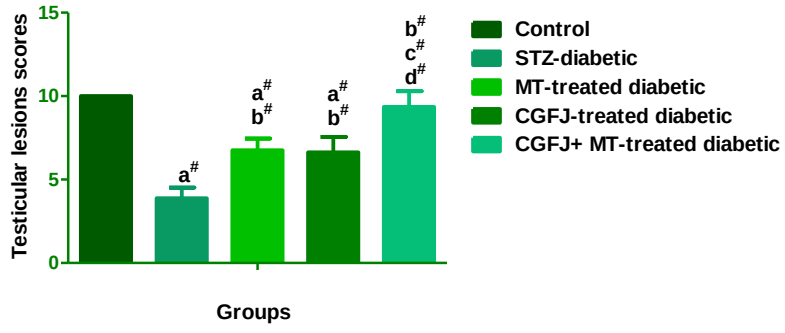

Fig. 5. Effect of CGMJ, MT, and CGMJ+MT on testicular lesions scores in diabetic rats. Data were presented as mean ± SD (8/each group). [a]Significant relative to control. [b]Significant relative to STZ-diabetic. [c]Significant relative to MT- treated diabetic. [d]Significant relative to CGMJ-treated diabetic. ([#]$p \leq 0.001$).

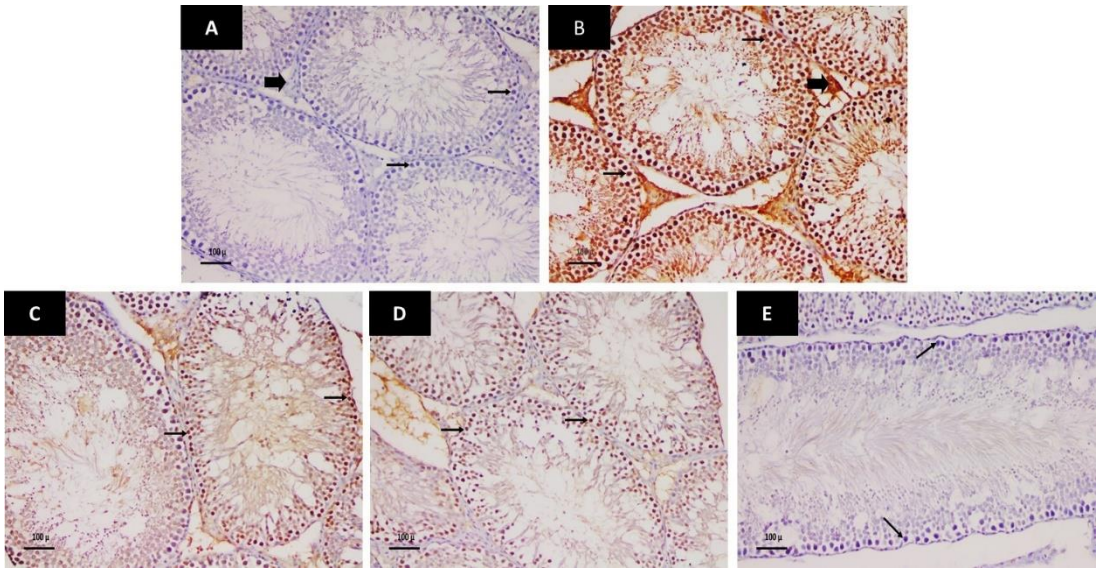

Fig. 6. Photomicrograph of seminiferous tubules of testis sections (NF-κB immunostaining with counterstain Haematoxylin, X 200). (A) Control group; (B) STZ-diabetic untreated group; (C) MT-treated STZ diabetic group; (D) CGFJ-treated STZ diabetic group; (E) CGFJ+MT-treated STZ diabetic group.

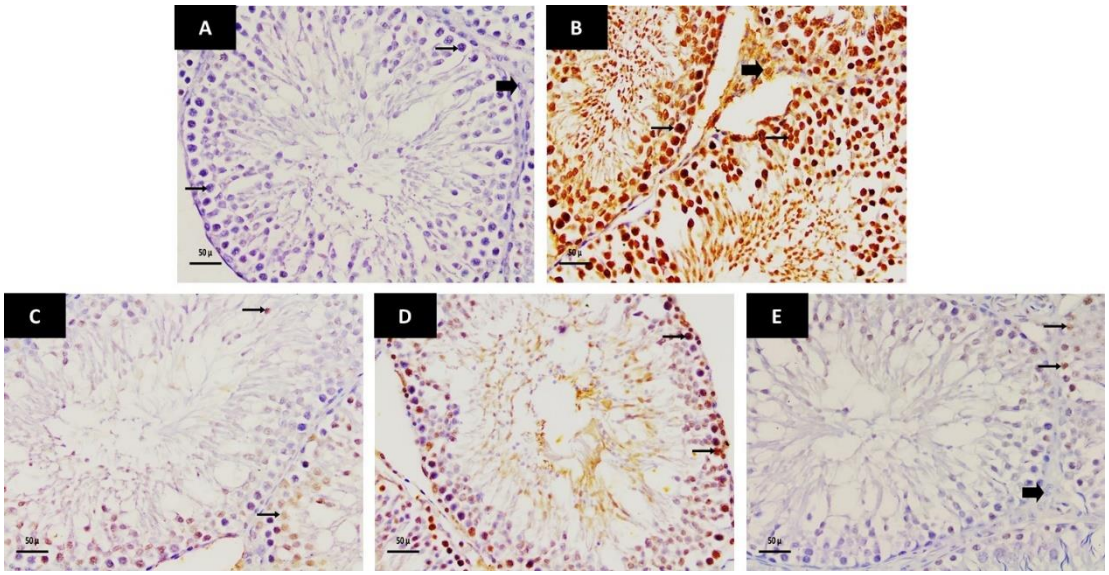

Fig. 7. Photomicrograph of seminiferous tubules of testis sections (NF-κB immunostaining with counterstain Haematoxylin, X 400). (A) Control group; (B) STZ-diabetic untreated group; (C) MT-treated STZ diabetic group; (D) CGFJ-treated STZ diabetic group; (E) CGFJ+MT-treated STZ diabetic group.

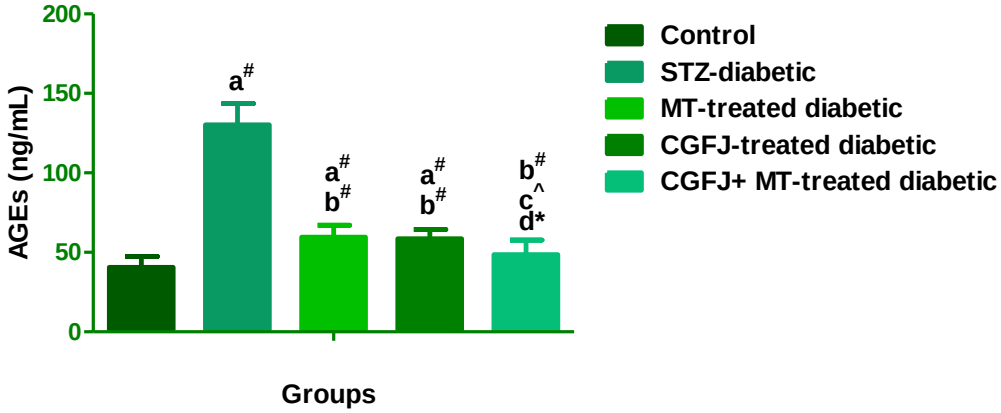

Fig 8. Effect of CGFJ, MT, and CGFJ+MT on serum AGEs in diabetic rats. Data were presented as mean ± SD (8/each group). [a] Significant relative to control. [b]Significant relative to STZ-diabetic. [c]Significant relative to MT-treated diabetic. [d]Significant relative to CGFJ-treated diabetic. ($^{\wedge}p \leq 0.05$, $^{*}p \leq 0.01$, and $^{\#}p \leq 0.001$).

References

1. Ogle, G. D., Wang, F., Haynes, A., Gregory, G. A., King, K. Deng, T. W., Dabelea, D., James, S., Jenkins, A. J., Li, X., Ma, R. C. W., Maahs, D. M., Oram, R. A., Pihoker, C., Svensson, J., Zhou, Z., Magliano, D. J. and Maniam, J. Global type 1 diabetes prevalence, incidence, and mortality estimates 2025: Results from the International Diabetes Federation Atlas, 11th Edition, and the T1D Index Version 3.0. *Diabetes Research and Clinical Practice*, **225**,112277 (2025).
2. Lu, X. and C. Zhao. Exercise and type 1 diabetes. *Advances in Experimental Medicine and Biology*, **1228**,107–21 (2020).
3. Jiang, Y., He, P., Sheng, K., Peng, Y., Wu, H., Qian, S., Xiaoling Guo, W.Ji. and Shan, X. The protective roles of eugenol on type 1 diabetes mellitus through NRF2-mediated oxidative stress pathway. *Elife*, **13**, RP96600. (2025).
4. Papadopoulou-Marketou, N., Paschou, S.A., Marketos, N., Adamidi, S., Adamidis, S. and Kanaka-Gantenbein, C. Diabetic nephropathy in type 1 diabetes. *Minerva Medica*, **109**(3), 218–28 (2018).
5. Zhu, J., Hu, Z., Luo, Y., Liu, Y., Luo, W., Du, X., Luo, Z., Hu, J., Peng, S., Vanamala, J. K.P.,Yorek, M. and Muramatsu. K. Diabetic peripheral neuropathy: pathogenetic mechanisms and treatment. *Frontiers in Endocrinology* **14**,1265372 (2024).
6. Wang, M., Li, Y., Li, S. and Lv, J. Endothelial dysfunction and diabetic cardiomyopathy. *Frontiers in Endocrinology*, **13**, 851941(2022).
7. Graziani, A., Scafa, R., Grande, G. and Ferlin, A. Diabetes and male fertility disorders. *Molecular Aspects of Medicine*, **99**,101303 (2024).
8. Tavares, R.S., Escada-Rebelo, S., Sousa, M.I., Silva, A., Ramalho-Santos, J. and Amaral, S. Can antidiabetic drugs improve male reproductive (dys)function associated with diabetes?. *Current Medicinal Chemistry*, **26**(22),4191–222 (2018).
9. Wagner, IV, Klöting, N., Savchuk, I., Eifler, L., Kulle, A., Kralisch-Jäcklein, S., Dötsch, J., Hiort, O., Svechnikov, K. and Söder, O. Diabetes type 1 negatively influences leydig cell function in rats, which is partially reversible by insulin treatment. *Endocrinology*, **162**(4),bqab017 (2021).
10. Maresch, C. C., Stute, D.C., Alves, M.G., Oliveira, P.F., de Kretser, D.M. and Linn. T. Diabetes-induced hyperglycemia impairs male reproductive function: A systematic review. *Human Reproduction Update*, **24**(1), 86–105 (2018).
11. Facondo, P., Di Lodovico, E., Delbarba, A., Anelli, V., Pezzaioli, L.C., Filippini, E., Cappelli, C., Corona, G. and Ferlin, A. The impact of diabetes mellitus type 1 on male fertility: systematic review and meta-analysis. *Andrology* **10**(3),426–40 (2022).
12. Prasad, C., Davis, K.E., Imrhan, V., Juma, S. and Vijayagopal. P. Advanced glycation end products and risks for chronic diseases: intervening through lifestyle modification. *American Journal of Lifestyle Medicine*, **13**(4),384–404 (2019).
13. Acharya, S. and Bhattacharyya. S. Dietary advanced glycation end-products (AGES) induced complications in type 1 diabetes mellitus. *Type 1 Diabetes - Causes, Treatments and Management : In: IntechOpen*. 7016 (2025). DOI: 10.5772/intechopen.1007016
14. Zhang, W., Tong, L., Jin, B. and Sun, D. Diabetic testicular dysfunction and spermatogenesis impairment: mechanisms and therapeutic prospects. *Frontiers in Endocrinology*, **16**, 1653975 (2025).
15. Zhu, J.L., Cai, Y.Q., Long, S.L., Chen, Z. and Mo, Z.C. The role of advanced glycation end products in human infertility. *Life Sciences*, **255**,117830 (2020).
16. Chen, Y., Zhang, Y., Ji, H., Ji, Y., Yang, J., Huang, J. and Sun, D. Involvement of hypoxia-inducible factor-1α in the oxidative stress induced by advanced glycation end products in murine leydig cells. *Toxicology in Vitro*, **32**, 146–53 (2016).
17. Raj, C.J., Aishwarya, C.V.S., Mounika, K.V.S., Mishra, B., Sumithra, B., Vishal, B. and Mandal, S.K. Deciphering the nexus between oxidative stress and spermatogenesis: a compendious overview. *Advances in Experimental Medicine and Biology*, **1391**,1–16(2022).

18. Tian, Y., Song, W., Xu, D., Chen, X., Li, X., Zhao, Y. and Zhao, Y. Autophagy induced by ros aggravates testis oxidative damage in diabetes via breaking the feedforward loop linking P62 and Nrf2. *Oxidative Medicine and Cellular Longevity*, **2020**,7156579 (2020).

19. Venditti, M., Romano, M.Z., Boccella, S., Haddadi, A., Biasi, A., Maione, S. and Minucci, S. Type 1 Diabetes impairs the activity of rat testicular somatic and germ cells through NRF2/NLRP3 pathway-mediated oxidative stress. *Frontiers in Endocrinology*, **15**, 1399256 (2024).

20. Agudelo-Sánchez, S., Mosquera-Palacios, Y., David-Úsuga, D., Cartagena-Montoya, S. and Duarte-Correa, Y. Effect of processing methods on the postharvest quality of Cape gooseberry (*Physalis peruviana* L.). *Horticulturae*, **9**(10), 1158 (2023).

21. Aboulthana, W.M., Omar, N.I., El-Feky, A.M., Hassan, A.K., Hasan, E.A., Seif, M. and Youssef, A.M. Phyto- and biochemical study on Cape gooseberry (*Physalis peruviana* L.) extract incorporated with metal nanoparticles against hepatic injury induced in rats. *Natural Product Research*, **2024**,1–14 (2024).

22. Pillai, J. R., Wali, A.F., Menezes, G.A., Rehman, M.U., Wani, T.A., Arafah, A., Zargar, S. and Mir, T.M. Chemical composition analysis, cytotoxic, antimicrobial and antioxidant activities of *Physalis angulata* L.: A comparative study of leaves and fruit. *Molecules* **27**(5),1480 (2022).

23. Fuente, F.P., Nocetti, D., Sacristán, C., Ruiz, P., Guerrero, J., Jorquera, G., Uribe, E., Bucarey, J.L., Espinosa. A. and Puente, L. *Physalis peruviana* L. pulp prevents liver inflammation and insulin resistance in skeletal muscles of diet-induced obese mice. *Nutrients*, **12**(3),700 (2020).

24. Abdel Moneim, A.E. Prevention of carbon tetrachloride (CCL4)-induced toxicity in testes of rats treated with *Physalis peruviana* L. fruit. *Toxicology and Industrial Health*, **32**(6), 1064–73 (2016).

25. Elgazar, A. F., Shalaby, M. A. and AboRaya. A. O. Potential anticancer effect of Cape gooseberry fruit juice on rats with induced-colon cancer. *The Scientific Journal of Specific Education and Applied Sciences*, **6**(15), 240–62 (2023).

26. Zakaria, R., Abelbaky, M.S. and Abo-Raya, A.O. Effect of golden berry (*Physalis peruviana*) fruits and its peels on acute hepatotoxicity with diabetic rats. *Egyptian Journal of Applied Science* **35**(11),158–71 (2020).

27. Shahein, M.R., Atwaa, E.H., Radwan, H.A., Elmeligy, A.A., Hafiz, A. A., Albrakati, A. and Elmahallawy, E.K. Production of a yogurt drink enriched with golden berry (*Physalis pubescens* L.) juice and its therapeutic effect on hepatitis in rats. *Fermentation*, **8**(3),112 (2022).

28. Zeng, F., Luo, J., Han, H., Xie, W., Wang, L., Han, R., Chen, H., Cai, Y., Huang, H. and Xia, Z. Allopurinol ameliorates liver injury in type 1 diabetic rats through activating Nrf2. *International Journal of Immunopathology and Pharmacology* **35**, 31416 (2021).

29. Furman, B. L. Streptozotocin-induced diabetic models in mice and rats. *Current Protocols*, **1**(4),e78(2021).

30. Animals, Guide for the Care and Use of Laboratory. Guide for the care and use of laboratory animals. *Guide for the Care and Use of Laboratory Animals* (2011).

31. Liu, Z., Yu, X., Tong, C. and Qi, H. Renal dysfunction in a mouse model of gdm is prevented by metformin through MAPKs. *Molecular Medicine Reports*, **49**(5), 4491–99 (2019).

32. Kinasih, L.S., Djamiatun, K. and Al-Baarri. A.N. Golden berry (*Physalis peruviana*) juice for reduction of blood glucose and amelioration of insulin resistance in diabetic rats. *Jurnal Gizi Dan Pangan*, **1** (15), 37–44 (2020).

33. Johnsen, S.G. Testicular biopsy score count-A method for registration of spermatogenesis in human testes: normal values and results in 335 hypogonadal males. *Hormone Research in Paediatrics*, **1**(1), 2–25 (1970).

34. Delen, O. and Uz, Y. H. Protective effect of pyrrolidine dithiocarbamate against methotrexate-induced testicular damage. *Human and Experimental Toxicology*, **40** (12 suppl), S164–77 (2021).

35. Shoorei, H., Khaki, A., Khaki, A.A., Hemmati, A.A., Moghimian, M. and Shokoohi, M. The ameliorative effect of carvacrol on oxidative stress and germ cell apoptosis in testicular tissue of adult diabetic rats. *Biomedicine & Pharmacotherapy*, **111,** 568–78 (2019).

36. Zhao, L., Zhao, J., Dong, Z., Xu, S., and Wang, D. Mechanisms underlying impaired spermatogenic function in orchitis induced by busulfan. *Reproductive Toxicology*, **115**, 1–7 (2023).

37. Gültekin, B., Çetinkaya Karabekir, S., Çinar Ayan, I., Savaş, H.B., Cüce, G. and Kalkan, S.S. Effect of carvacrol on diabetes-induced oxidative stress, fibrosis and apoptosis in testicular tissues of adult rats. *Physiological Research*, **74**(3),459–69 (2025).

38. Pereira, S. C., Oliveira, P.F., Oliveira, S.R., Pereira, M.L. and Alves, M.G. Impact of environmental and lifestyle use of chromium on male fertility: focus on antioxidant activity and oxidative stress. *Antioxidants*, **10**(9),1365(2021).

39. Nna, V.U., Bakar, A.B.A., Ahmad, A., Eleazu, C.O. and Mohamed, M. Oxidative stress, NF-KB-mediated inflammation and apoptosis in the testes of streptozotocin–induced diabetic rats: combined protective effects of malaysian propolis and metformin. *Antioxidants*, **8**(10), 465(2019).

40. Nna, V.U., Bakar, A.B.A., Ahmad, A. and Mohamed, M. Diabetes-Induced Testicular Oxidative Stress, Inflammation, and Caspase-Dependent Apoptosis: The Protective Role of Metformin. *Archives of Physiology and Biochemistry*, **126**(5), 377–88 (2020).

41. Barnabei, L., Laplantine, E., Mbongo, W., Rieux-Laucat, F., and Weil, R. NF-KB: At the borders of autoimmunity and inflammation. *Frontiers in Immunology*, **12**, 716469 (2021).

42. Simó-Servat, O., Planas, A., Ciudin, A., Simó, R. and Hernández, C. Assessment of advanced glycation end-products as a biomarker of diabetic outcomes. *Endocrinologia, Diabetes y Nutricion*, **65(**9), 540–45 (2018).

43. Dutta, S., Majzoub, A. and Agarwal, A. Oxidative stress and sperm function: a systematic review on evaluation and management. *Arab Journal of Urology*, **17**(2),87–97 (2019).

44. Mori, Y., Terasaki, M., Osaka, N., Fujikawa, T., Yashima, H., Saito, T., Kataoka, Y., Ohara, M., Higashimoto, Y., Matsui, T. and Yamagishi, S.I. DNA aptamer raised against advanced glycation end products improves sperm concentration, motility, and viability by suppressing receptors for advanced glycation end product-induced oxidative stress and inflammation in the testes of diabetic mice. *International Journal of Molecular Sciences*, **25**(11), 5947(2024).

45. Carvalho, R.P., Guimarães-Ervilha,L.O., Viana, A.G., Ribeiro, I.M., Ramirez-Lopez, C. and Machado-Neves, M. A systematic review and meta-analysis of the effects of diabetes on human and murine epididymis. *Andrology*, **13**(3),70026(2025).

46. Othman, M.S., Nada, A., Zaki, H. S. and Abdel Moneim, A. E. Effect of *Physalis peruviana* L. on cadmium-induced testicular toxicity in rats. *Biological Trace Element Research*, **159**(1–3), 278–87 (2014).

47. Ángel-Martín, A., Vaillant, F. and Moreno-Castellanos, N. Daily consumption of golden berry (*Physalis peruviana*) has been shown to halt the progression of insulin resistance and obesity in obese rats with metabolic syndrome. *Nutrients*, **16**(3),30365 (2024).

48. Muzaffar, K., Sofi, S.A., Makroo, H.A., Majid, D. and Dar, B. N. Insight about the biochemical composition, postharvest processing, therapeutic potential of indian gooseberry (Amla), and its utilization in development of functional foods- A comprehensive review. *Journal of Food Biochemistry*, **46**(11),14132 (2022).

49. Singh, A. Herbal-based nutraceuticals in management of lifestyle diseases: experience from indian population. *Future Integrative Medicine*, **3**(2),106–115 (2024).

50. Akhtar, M.S., Ramzan, A., Ali, A. and Ahmad, M. Effect of Amla fruit (*Emblica officinalis gaertn*.) on blood glucose and lipid profile of normal subjects and type 2 diabetic patients. *International Journal of Food Sciences and Nutrition*, **62**(6), 609–16 (2011).

51. Nitcheu Ngemakwe, P.H., Remize, F., Thaoge, M. L., and Sivakumar, D. Phytochemical and nutritional properties of underutilised fruits in the southern african region. *South African Journal of Botany*, **113**,137– 49 (2017).

52. Petkova, N.T., Popova, V.T., Ivanova, T.A., Mazova, N.N., Panayotov, N.D. and Stoyanova, A. Nutritional composition of different Cape gooseberry genotypes (*Physalis peruviana* l.) - A comparative study. *Food Research*, **5**(4), 191–202 (2021).

53. Aljadani, H. M., Shehta, H.A., Khattab, H.A.H., and El-Shitany, N. A. Protective effects of golden berry (*Physalis peruviana* l.) juice against diabetic renal injury in rats. *Egyptian Journal of Veterinary Sciences*, **56**(6), 1195–206 (2025).

54. Bhatia, J., Bhatia, J., Malik, S. and Suchal, K. A17490 kaempferol ameliorates myocardial injury in diabetic rats by suppressing oxidative stress, inflammation and apoptosis. *Journal of Hypertension*, **36**,e89 (2018).

55. Yang, B.N., Choi, E.H., and Shim, S.M. Inhibitory activities of kaempferol against methylglyoxal formation, intermediate of advanced glycation end products. *Applied Biological Chemistry*, **60**(1), 57–62 (2017).

56. Alshehri, A.S., El-Kott, A.F., El-Kenawy, A. E., Zaki, M.S.A., Morsy, K., Ghanem, R. A., Salem, E.T., Ebealy, E. R., Khalifa, H.S., Altyar, A. E., AlGwaiz, H.I.M., Ibrahim, E. H., Mahmoud, M. S., Dallak, M. A. and Abd-Ella, E. M. The ameliorative effect of kaempferol against CdCl2-mediated renal damage entails activation of Nrf2 and inhibition of NF-KB. *Environmental Science and Pollution Research*, **29**(38),57591–602 (2022).

عصير فاكهة الحرنكش يُعالج تلف الخصية لدى الفئران المصابة بداء السكري من النوع الأول: استهداف تثبيط النواتج النهائية لتسكر بروتينات الدم المتقدمة، الإجهاد التأكسدي، والالتهابات

صفية محمد عبد الله باحشوان[1]، هيا محمد الجدعاني[2]، أفنان حسن ساعاتي[2]، إيمان عباس علي عبد الجواد[2]، منال محمود صدقة منصوري[2]*، صالح عابد الحربي[3]، أحلام عبد العزيز الأحمدي[4] وسعاد شاكر علي[5]

[1] قسم علوم الأحياء، جامعة الملك عبد العزيز، فرع رابغ، المملكة العربية السعودية.
[2] قسم الغذاء والتغذية، كلية العلوم الإنسانية والتصاميم، جامعة الملك عبد العزيز، جدة، المملكة العربية السعودية.
[3] قسم المختبرات، خدمات إرادة، التجمع الصحي الثاني، مجمع إرادة للصحة النفسية، وزارة الصحة، جدة، المملكة العربية السعودية.
[4] قسم العلوم البيولوجية، كلية العلوم، جامعة الملك عبد العزيز، جدة، المملكة العربية السعودية.
[5] قسم علم الأنسجة وبيولوجيا الخلية، كلية الطب، جامعة أسيوط، عضو باحث في يوسف عبد اللطيف جميل.

الملخص

الأمراض الأيضية، مثل داء السكري من النوع الأول، ترتبط غالبا بانخفاض الخصوبة وتلف الخصية عند الذكور، الآليات المسببة لذلك، ترجع الي أن ارتفاع مستويات جلوكوز الدم، قد تؤدي إلى خلل في مسار الغدة النخامية – الخصية، الميتوكوندريا، الالتهابات المزمنة، الإجهاد التأكسدي، وتراكم النواتج النهائية لتسكر بروتينات الدم المتقدمة. التأثير المؤكد للإجهاد التأكسدي على الخصوبة أدي الي القاء الضوء على استخدام مضادات للأكسدة للحماية من تلف الخصية الناجم عن السكري. يعزز عصير الحرنكش أنظمة الدفاع المضادة للأكسدة ضد السمية التناسلية. هدفت هذه الدراسة إلى تقييم التأثير الوقائي المُحتمل لعصير الحرنكش ضد تلف الخصية المرتبط بـالسكري النوع الأول في الفئران، بالإضافة الي تقييم تأثيره على النواتج النهائية لتسكر بروتينات الدم المتقدمة ومؤشرات الإجهاد التأكسدي. تم تقسيم الفئران الذكور عشوائيًا الي خمس مجموعات كالتالي: الضابطة؛ مصابة بالسكري؛ مصابة بالسكري ومعالجه بالميتافورمين؛ مصابه بالسكري ومعالجه بعصير الحرنكش؛ ومصابة بالسكري ومعالجة بالميتافورمين وعصير الحرنكش. تم احداث السكري النوع الأول بحقن الاستربتوزيتوسين (٦٥ ملجم/كجم) جرعة واحدة في الغشاء البروتيوني .أُعطي الميتافورمين (٦٠٠ ملجم/كجم) وعصير الحرنكش (٥ مل/كجم) فمويا جرعة واحدة يوميًا لمدة ٧٠ يومًا .أظهرت النتائج أن عصير الحرنكش خفض مستوى الجلوكوز ورفع مستويات الأنسولين وهرمون التستوستيرون في المصل، وحسن هرمونات الغدد التناسلية. حسن من مستويات الإجهاد التأكسدي، كما يتضح من انخفاض مستويات المالوندالدهيد، وارتفاع نشاط الأنزيمات المضادة للأكسدة . بالإضافة إلى ذلك، قد أحدث انخفاض في مستويات النواتج النهائية لتسكر بروتينات الدم المتقدمة وتغيير في NF-κB. أظهر عصير الحرتكش تحسناً بدرجة معنوية تأثير الميتافورمين، حيث خفف من تلف الخصية الناتج عن السكري النوع الأول من خلال تثبيط النواتج النهائية لتسكر بروتينات الدم المتقدمة، الالتهاب، والإجهاد التأكسدي.

الكلمات الدالة: النواتج النهائية لتسكر بروتينات الدم المتقدمة، الحرنكش، مرض السكري النوع الأول، الالتهابات، الإجهاد التأكسدي.