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The Effect of Broccoli (*Brassica oleracea*) in Oxidative Stress Status of Obese Diabetic Rats

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Abstract

The present work aimed to investigate the possible antioxidant and hypoglycemic effects of broccoli in oxidative stress of obese diabetic rats. Thirty adult male Sprague-Dawley rats were divided randomly into six equal groups (n=5) as follow: Group 1: negative control, was fed on a basal diet. Groups 2-6 were fed on HFD for 6 weeks to induce obesity. The obese rats were then rendered diabetic by subcutaneous injection with a single dose of alloxan (120 mg/kg). Group 2 was kept as a positive control group (obese diabetic rats) and groups 3, 4, 5 and 6 fed on HFDs-alloxan induced diabetic and supplemented with 2.5, 5, 7.5 and 10% dried broccoli. At the end of the experiment, rats were scarified and serum was collected for biochemical analyses. The administration of HFD-alloxan resulted in significant elevations in body weight gain, feed efficiency ratio, peritoneal fat pad, serum total cholesterol, triglyceride, LDL-c, VLDL-c, leptin, glucose, insulin, interleukin-1 beta, malondialdehyde, AST, ALT, ALP, urea, uric acid and creatinine levels in a positive control group as compared to negative control, and levels of serum HDL-c and glutathione peroxidase were significantly decreased. On the other hand, a supplemented diet with dried broccoli powder attenuated these adverse effects and biochemical alterations that caused by HFD-alloxan administration. In conclusion, broccoli exhibits an antioxidant and hypolipidemic activity and effective in reducing glucose level in obese diabetic rats. The study recommends that intake of broccoli may be beneficial for patients who suffer from diabetes and obesity.

Keywords: Broccoli, Oxidative stress, Obesity, Diabetes, High fat diet, Rats.

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1. Introduction

Dietary habits are of great importance in human health. Diabetes mellitus (DM) is a serious global health problem, particularly increasing in the Middle East. This development is due to the extensive adaptation in lifestyle, including the tendency to a westernized diet, less physical activity, obesity and smoking [1]. Diabetes mellitus is a group of disorders characterized by chronic hyperglycemia due to a defect in the body's ability to convert glucose (sugar) to energy. It is a common endocrine disorder that involves dysfunction of pancreatic β cell. The loss of β cell mass and the progressive decline in β cell function are an early profile of diabetes [2-3]. Moreover, diabetes induces changes in the tissue content and activity of the antioxidant enzymes [4]. Prolonged hyperglycemia and hyperlipidemia are associated with oxidative stress which is presently seen as an important piece of the puzzle for understanding the origin, development, and pathogenesis of type 1 diabetes and type 2 diabetes and other adverse effects including diabetic complications such as nephropathy, neuropathy, cardiovascular and liver diseases.

Moreover, sustained oxidative stress triggers the expression of different inflammation-regulated genes and activates pathogenic proinflammatory pathways that also contribute to a variety of diabetic complications [5]. Reactive oxidative (ROS) and reactive nitrogen species (RNS) are reactive, short lived and very unstable chemical entities that are produced as a result of normal physiological processes in cells and play important roles in cellular signaling, gene transcription and the immune response [6]. Broccoli (*Brassica oleracea*) is a member of the Brassicaceae family which is rich in potential health boosting components like vitamins, minerals, dietary fiber, flavonol glycosides, hydroxycinnamic acids, and Sulphur containing compounds such as the glucosinolates [7-8]. Broccoli is also rich in vitamin C, a major antioxidant in Brassica vegetables [9-10]. Therefore, this study was conducted to evaluate the possible antioxidant and hypo-glycaemic effects of broccoli in oxidative stress of obese diabetic rats.

2. Materials and Methods

2.1. Materials

Fresh broccoli was purchased from a local market in Egypt. Biochemical kits, casein, cellulose, choline chloride, D-L methionine, vitamins and mineral constituents were purchased from El-Gomhoriya Pharmaceutical Company, Cairo, Egypt. Starch, soy oil, and sucrose were obtained from the Egyptian local market. Thirty adult male albino rats (Sprague Dawley strain), weighing about 200 ±10 g b.wt. were obtained from the Laboratory Animal Colony, Agricultural Research Center, Giza, Egypt. Alloxan monohydrate was purchased from Kemet Medical Company, Cairo, Egypt.

2.2. Methods

2.2.1. Preparation of Dried Broccoli

Broccoli was washed, sliced into small pieces, and oven-dried at 50°C [11]. Dried Broccoli was grounded using a grinder into a fine powder till used for both chemical composition and isolation phenolic compounds and for the preparation of a supplemented diet. The preparation of dried broccoli was conducted at the National Research Center.

2.2.2. Chemical Analysis of Broccoli

The chemical analysis of broccoli was conducted in the Food Analysis Unit, Agricultural Research Center, Egypt.

- Chemical composition (carbohydrates, protein, fats, moisture and ash) was determined according to Golay et al., [12].
- 1,1-diphenyl-2-picrylhydrazyl (DPPH) radical-scavenging activity was evaluated according to Brand-Williams [13].
- Total phenolic compounds were determined by Spectrophotometer according to Goupy et al., [14].

2.2.3. Induction of Obesity and Diabetes

Obesity and acute hyperlipidemia were induced by feeding rats on high-fat diet (HFD) containing (saturated fat 19%, soybean oil 1% to provide essential fatty acids, sucrose 10%, casein 20%, cellulose 5%, vitamin mixture 1%, salt mixture 3.5%, choline chloride 0.25% and the remainder is corn starch) for 4 weeks to induce obesity in rats [15]. A 3-6 weeks HFD feeding is sufficient to induce obesity, and this model of obese rats is closely resembles the reality of obesity in humans [16]. The obese rats were then rendered diabetic by subcutaneous injection with a single dose of alloxan (120 mg/kg) to induce diabetes [17]. Induction of obesity and diabetes of rats was conducted at the Research Labs, Agricultural Research Center, Giza, Egypt.

2.2.4. Diet Preparation and Experimental Animal Design

The basal diet was prepared according to the AIN-93M diet [18]. Thirty adult male albino rats were housed in well conditions and fed on basal diet in Research Labs, Agricultural Research Center, Giza, Egypt. After one week of acclimatization, the rats were randomly divided into two main group as follows:

- **First group:** Negative control group, rats (n=5) fed on a basal diet only during the experimental period.
- **Second group:** Rats (n=25) were fed on HFD for 6 weeks to induce obesity. After six weeks, the obese rats were then rendered diabetic by subcutaneous injection with a single dose of alloxan (120 mg/kg). After 72 h of injection, fasting blood glucose level was measured. Blood glucose measurement was performed on tail-vein blood. The animals that did not develop more than 200 mg/dL glucose levels were omitted from the study. Rats were divided to five subgroups as follow:

i. **Subgroup (1):** Rats (served as a positive control group or obese diabetic rats) were fed on HFD, alloxan - induced diabetic.
ii. **Subgroup (2):** Rats were fed on HFD, alloxan - induced diabetic and supplemented with 2.5% dried broccoli powder (25g/kg HFD).
iii. **Subgroup (3):** Rats were fed on HFD, alloxan - induced diabetic and supplemented with 5% dried broccoli powder (50g/kg HFD).
iv. **Subgroup (4):** Rats were fed on HFD, alloxan - induced diabetic and supplemented with 7.5% dried broccoli powder (75g/kg HFD).
v. **Subgroup (5):** Rats were fed on HFD, alloxan - induced diabetic and supplemented with 10% dried broccoli powder (100g/kg HFD).

During the experiment period, the quantities of diet, which were consumed and/or waste, were recorded every day. Water and basal diet had introduced under hygienic conditions. At the end of the feeding trial (10 weeks) rats were fasted over-night being scarified and was collected, then centrifuged to obtain serum for biochemical analysis.

2.2.5. Biological Evaluation

Feed intake was recorded daily and animals were weighed at the beginning and twice a week throughout the experimental period. Body weight gain percent (BWG%) and feed efficiency ratio were determined according to Chapman et al., using the following equation [19]:

$$BWG\% = \frac{Final\ body\ weight - Initial\ body\ weight}{Initial\ body\ weight} \times 100$$

$$FER = Weight\ gain\ /\ Feed\ intake.$$

2.2.5.1. Peritoneal fat pad% (PFD %)

At the end of the experimental period (the last 4 weeks), the percentage of peritoneal fat pad was calculated as follows:

$$PFD = \frac{Weight\ of\ peritoneal\ fat\ pad}{Weight\ of\ rat} X\ 100$$

2.2.6. Biochemical Analysis of Serum

Serum glucose, leptin and insulin levels were determined according to the methods described by Trinder et al., Zhang et al., and Hold et al., respectively [20-22].

Serum total cholesterol, triglyceride and high-density lipoprotein cholesterol were determined according to Richmond et al., Wahlefeld et al., and Albers et al., respectively [23-25]. Low density lipoprotein cholesterol and very low-density lipoprotein cholesterol were calculated according to Friedewald et al., [26]. Serum aspartate aminotransaminase (AST), alanine aminotransaminase (ALT), urea and creatinine were determined according to the method described by Young et al., [27]. Serum alkaline phosphates (ALP) and uric acid were determined according to Roy et al., and Milena et al., respectively [28-29]. Interleukin-1 beta, malondialdehyde and glutathione peroxidase were determined according to Dinarello et al., Draper and Hadley and Hissin and Hilf respectively [20,31-32].

2.3. Statistical Analysis

Results were expressed as the mean standard error ± SE. Data were statistically analyzed for variance using the "ANOVA" test at $P \leq (0.05)$ using SPSS statistical software, version 20 according to Armitage and Berry [33].

3. Results and Discussion

3.1. Chemical composition, total polyphenols, and antioxidant capacity of broccoli florets

Results in (Table 1) showed that the chemical composition of broccoli. Broccoli contained protein, carbohydrate, fiber, moisture, ash and fat with values 33.2%, 30.64%, 13.42%, 10.2%, 8.78% and 3.76%, respectively. These results are in agreement with U.S.D.A. which concluded that the content of broccoli from moisture was (89.30), protein (2.82), fat (0.37), carbohydrate (6.64) and fiber (2.6) [34]. In another study, Sigmond et al., showed carbohydrate, fiber, ash, moisture, protein and fat of broccoli were 53.62, 11.00, 2.68, 2.40, 24.50 and 5.5%, respectively [35]. In addition to data in Table 1, revealed that broccoli contained 12157 ppm (Gallic acid equivalent) of total phenols and 3.58 mg/ml of DPPH. These findings approved with results in the study of Campas-Baypoli et al., indicated Total polyphenols (17.21 mg Gallic acid equivalent /g dry matter) and Antioxidant capacity (mmol TE/g dry matter) such as, DPPH Radical scavenging capacity (112) [36]. A strong link between consumption of fruits and vegetables and their health benefits are due to their high nutritional value and functional components with antioxidant properties. Antioxidants are compounds which control and scavenge oxidative damage in foods and bio-molecules by slowing or inhibiting the oxidative process caused by ROS, thus enhancing the quality of the products [37]. Results in (Table 2) showed the effect of broccoli on feed intake (FI), body weight gain%, (BWG%) feed efficiency ratio (FER) and peritoneal fat pad% (PFP%) of obese diabetic rats. Results showed that feed intake decreased in positive control rats compared with the negative control rats. On the other hand, BWG%, FER and PFP% significantly increased ($P \leq 0.05$) in +ve control group when compared to -ve control group. Supplementation with broccoli on obese diabetic rats caused a reduction in the feed intake, BWG, FER and PFP% when compared with the +ve control group. These results agreement with Zhang et al., who noticed HFD-alloxan led to a significant increase in body weight in the experimental mice [38].

Moreover, Aranaz et al., reported that after 10 weeks of broccoli supplementation, animals exhibited reduced body weight gain and feed efficiency, retroperitoneal fat mass and adipocyte size [39]. In another study showed that broccoli extract (florets and stalks), administered *per os* at a dose of 200 or 400 mg/kg/day for 1-month, reduced body weight gain and adipose tissue index in rats fed a HFD [40]. Data in (Table 3) revealed that positive control rats had a significant ($P \leq 0.05$) increases in serum levels of TC, TG and LDL-c, VLDL-c and a significant decrease ($P \leq 0.05$) in HDL-c when compared to negative control group. Obese diabetic rats that treated with broccoli had significant ($P \leq 0.05$) reduction in the elevated serum TC, TG, LDL-c and VLDL-c levels and an increase in serum HDL-c when compared with positive control group. The mice with HFD-alloxan-induced diabetes showed abnormalities in lipid metabolism, as evidenced by increased TG, TC and LDL-C levels and decreased HDL-C levels, similar to the characteristics of human type 2 diabetes [41-42]. Hypertriglyceridemia may occur due to increased absorption and formation of triglycerides in the form of chylomicrons following consumption of a diet rich in fat or through increased endogenous production of TG-enriched hepatic VLDL and decreased TG uptake in peripheral tissues [43]. These findings were confirmed by Ranaweera et al., who found that the treatment with broccoli extract significantly reduced ($P \leq 0.05$) TG content, LDL-c and total cholesterol [44]. On the other hand, both animal and interventional studies have reported that broccoli consumption could contribute to reduce cholesterol blood concentration [45-46]. In an animal model, supplementation with 200 and 400 mg/kg of a broccoli extract reduced the levels of TG, total cholesterol, LDL-cholesterol and increased HDL [40]. Table 4 indicated that the higher level in serum leptin, glucose and insulin in obese diabetic rats group compared control group. Supplementation with broccoli was diminished the increasing in levels of leptin, glucose and insulin in treated group compared with control group. The effects of obesity-promoted increased leptin levels are often insufficient to mitigate increased body fat, potentially because individuals with overweight or obesity may be susceptible to "leptin resistance" [47]. That said, it is more likely that the failure of physiologic, counter-regulatory mechanisms to prevent excessive body fat gain (e.g., release of leptin from hypertrophied adipocytes) is because other physiologic and environmental promoters of obesity overwhelm leptin's anti-obesity effects [48]. Leptin may have mixed effects on glucose metabolism. Leptin may decrease insulin secretion and may contribute to insulin resistance. For example, chronic leptin stimulation of the arcuate nucleus of the hypothalamus may promote protein tyrosine phosphatase 1B (PTP1B), which inhibits insulin activity [49]. Increased leptin and insulin levels (along with postprandial effects) increase sympathetic nervous system activity, potentially contributing to insulin resistance. Conversely, leptin may increase glucose tissue uptake by muscle and brown adipose tissue, decrease glucagon secretion by the pancreas, decrease corticosterone by the adrenal gland, decrease lipolysis in white adipocytes, and decrease gluconeogenesis and glucose output by the liver [50-52]. These findings agreed with Miao et al., and Wang et al.

They found that hypoglycemic effect of dried broccoli may be due to the active component sulforaphane, a natural compound present in broccoli with many promising health benefits [53-54]. Previous studies performed in diabetic animal models and diabetic populations have proposed broccoli as a functional food with nutritional and therapeutic values against insulin resistance [55-58]. Broccoli has been previously proposed as an effective supplement for improving glycaemic control. Interventional studies have suggested that the supplementation with broccoli (extracts or powder) could improve glucose tolerance and insulin sensitivity [59-60]. The effect of broccoli on glutathione peroxidase (GPx) activity, malondialdehyde (MDA) and interleukin-1 beta (IL-1β) of obese diabetic rats were recorded in (Table 5). Results showed that the positive control group had a significant reduction ($P \leq 0.05$) in level of GPx activity while had a significant ($P \leq 0.05$) elevation in serum IL-1β and MDA concentrations when compared with the negative control group. It was observed that, there was a significant increase ($P \leq 0.05$) in GPx level and decrease in IL-1β and MDA content for obese diabetic rats that treated with broccoli when compared to positive control group. Results of the present study were in the same line with Mohammed et al., who reported that broccoli caused increase in the levels of superoxide dismutase (SOD) and total glutathione and caused reduction in the level of MDA when compared to the injected group [61]. On the other hand, Sharma and Sangha revealed that the oxidative stress biomarkers such as, SOD and GPx activity were improved with broccoli supplementation [62]. The total antioxidant activity was also significantly increased in all the treated rats with broccoli while significantly reduced lipid peroxidation levels as compared to positive control group. TNF-α and IL-1β are members of a group of cytokines that involved in systemic inflammation, act synergistically and stimulate acute phase reaction. They are produced mainly by macrophages, but a broad variety of other cell types is also involved in their production. They are inducers of endothelial adhesion molecules, which are essential for the adhesion of leukocytes to the endothelial surface prior to their migration into the tissues [63]. They also augment neutrophil–derived superoxide generation, leading to oxygen radical-mediated tissue damage. Stimulation of intestinal epithelial cells with TNF-α has also induced apoptosis [64-65]. Previous studies showed a significant increase in proinflammatory cytokines in response to Cis [66-67]. Epidemiological studies have shown that the consumption of fruits and vegetables could control the morbidity and mortality rates of certain types of diseases. This is due to the presence of bioactive components, namely fibers, polyphenol compounds, flavonoids, isoflavones, tocopherols and ascorbic acid [68-69]. These positive effects could be due to the dietary antioxidants which play a significant role in protecting against reactive oxygen species [70]. Broccoli constitutes a good candidate of functional food, based on its high content in glucosinolates that can be hydrolyzed to the lipophilic isothiocyanates, with reported benefits against certain forms of cancer, as well as with antioxidant activity [59,71,73]. Studies have confirmed the synergistic effect of dietary antioxidants with cellular reductants in scavenging free-radicals and chelating transition metals that are catalysts in lipid peroxidation [74].

On the other hand, administration of broccoli extracts restored GSH and TAC. These effects may be due to its hypoglycaemic and free radical scavenging properties based on the fact that broccoli contains several compounds such as total polyphenol, flavonoids, and other components that can act as a potent antioxidant [75]. Results in (Table 6) showed that serum concentration of AST, ALT and ALP significantly increased ($P \leq 0.05$) in obese diabetic control group when compared to negative control group. Rats that fed on broccoli flower powder had a significant reduction ($P \leq 0.05$) in serum levels of AST, ALT and ALP when compared to positive control group, as recorded in Table 4. These results were the same line with the results of Zhang et al., who concluded the diabetic hyperglycemia induced by HFD-alloxan treatment produced elevated levels of AST and ALT in the serum, which are considered highly indicative of liver dysfunction [76]. Epidemiological studies show that diabetic patients are at higher risk of developing chronic liver diseases and hepatocellular carcinoma. In experimental models of diabetes, both a high-carbohydrate/high-fat diet and alloxan exert their toxic effects on the liver and other organs in addition to pancreatic β-cells. The insulin insufficiency and hyperglycemia that result from β-cell necrosis further augment liver damage through reactive free radical mediated lipid peroxidation of hepatocellular membrane [77-78]. These findings are supported by Robbins et al., and Yoshida et al., who reported that cruciferous vegetables like broccoli may increase livers natural detoxification enzymes which protect it from damage and improve blood levels of liver enzymes [79-80]. Also, Abdalraheem and Salam, who observed that broccoli which helps the liver to produce detoxifying enzymes, they also contain sulfur compounds that aid with liver health [81]. In Al- Howiriny et al., study, results showed that the treatment of broccoli extract at the doses of 150 and 300 mg/kg produced significant hepatoprotection by reducing the activity of serum enzymes (ALT, AST and ALP) compared to the injured groups injected with CCl_4 [82]. Data demonstrated the effect of broccoli on serum kidneys functions of obese diabetic rats. Results showed that serum concentration of urea, creatinine and uric acid significantly increased ($P \leq 0.05$) in obese diabetic control group when compared to negative control group. Rats that fed on broccoli flower powder had a significant reduction ($P \leq 0.05$) in serum levels of urea, creatinine and uric acid when compared to positive control group in (Table 7). Hassan and Khedr concluded that the treatment with broccoli improvement serum lipid profiles, blood sugar, liver and kidney function (urea, uric acid and createnine) [83-84]. Also, Raeeszadeh et al., explored treatment of broccoli diminished the elevation in serum creatinine and urea levels compared with group received lead acetate [85]. Kidney removes metabolic wastes such as urea, uric acid, and creatinine. The concentrations of the metabolites increase in blood during renal diseases or renal damage may due to high activities of xanthine oxidase, lipid peroxidation, and increased triacylglycerol and cholesterol levels [86].

Table 1: Chemical composition, Total Polyphenols and Antioxidant Capacity of Broccoli.

Nutrient	Amount
Moisture %	10.2
Protein %	33.2
Fat %	3.76
Carbohydrated, by difference%	30.64
Fiber %	13.42
Ash %	8.78
Total polyphenols (ppm galic acid equivalent)	12157
Antioxidant Capacity of broccoli	
DPPH (mg/ ml)	3.58

Table 2: Effect of Broccoli on Feed Intake, Body Weight Gain%, Feed Efficiency Ratio and Peritoneal Fat Pad% of Obese Diabetic Rats.

Parameters / Groups	FI (g/d/rat)	BWG%	FER	PFP
G1: -Ve Control	21 ± 0.1^{ab}	23.15 ± 1.04^{ab}	0.085 ± 0.001^{b}	4.64 ± 0.18^{d}
G2: +Ve Control (obese diabetic rats)	20 ± 0.12^{c}	35.52 ± 1.77^{a}	0.129 ± 0.001^{a}	9.23 ± 0.38^{a}
G3: 2.5% DBP	21.5 ± 0.11^{b}	23.83 ± 1.85^{b}	0.081 ± 0.001^{b}	8.98 ± 0.29^{a}
G4: 5% DBP	22 ± 0.12^{a}	21.21 ± 1.54^{bc}	0.070 ± 0.001^{c}	8.51 ± 0.45^{ab}
G5: 7.5% DBP	23.5 ± 0.13^{a}	16.33 ± 0.85^{d}	0.053 ± 0.001^{d}	7.53 ± 0.32^{bc}
G6: 10% DBP	24 ± 0.15^{a}	12.21 ± 0.68^{e}	0.038 ± 0.001^{e}	6.68 ± 0.56^{c}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at $P\leq0.05$.
*** DBP =** Dried Broccoli Powder.

Table 3: Effect of Broccoli on Serum Lipid Profile of Obese Diabetic Rats.

Parameters / Groups	Total Cholesterol	Triglycerides	HDL-C	LDL-C	VLDL-C
	mg/dl				
G1: -Ve Control	170.27±0.91^{e}	137.59±4.51^{d}	60.49±1.17^{a}	82.26±1.00^{e}	27.52±0.90^{d}
G2: +Ve Control (obese diabetic rats)	236.71±1.65^{a}	183.96±3.83^{a}	36.08±0.93^{d}	163.84±1.16^{a}	36.79±0.76^{a}
G3: 2.5% DBP	214.45±3.49^{b}	165.19±2.33^{b}	55.07±0.84^{b}	126.33±3.60^{b}	33.04±0.46^{b}
G4: 5% DBP	191.05±3.32^{c}	152.29±1.77^{bc}	54.52±1.34^{bc}	106.07±2.37^{c}	30.45±0.35^{bc}
G5: 7.5% DBP	178.28±2.85^{d}	147.24±1.17^{cd}	52.18±1.08^{c}	96.65±1.66^{d}	29.44±0.23^{bc}
G6: 10% DBP	173.65±1.90^{de}	145.36±1.66^{cd}	51.08±1.67^{c}	93.50±0.45^{d}	29.07±0.33^{cd}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at P≤0.05.
*** DBP =** Dried Broccoli Powder.

Table 4: Effect of Broccoli on Serum Leptin, Glucose and Insulin of Obese Diabetic Rats.

Parameters / Groups	Leptin	Glucose	Insulin
	ng/ml	mg/dl	ng/ml
G1: -Ve Control	8.08±0.11^{d}	92.82±1.01^{e}	3.69±0.10^{e}
G2: +Ve Control (obese diabetic rats)	16.76±0.22^{a}	241.70±0.88^{a}	6.36±0.13^{a}
G3: 2.5% DBP	15.03±0.20^{b}	201.19±0.90^{b}	5.70±0.08^{b}
G4: 5% DBP	14.25±0.27^{b}	167.62±0.87^{c}	5.10±0.05^{c}
G5: 7.5% DBP	9.81±0.13^{c}	101.77±0.74^{d}	4.59±0.08^{d}
G6: 10% DBP	9.04±0.30^{cd}	98.33±0.65^{de}	4.12±0.03^{d}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at P≤0.05.
*** DBP =** Dried Broccoli Powder.

Table 5: Effect of Broccoli on Serum Glutathione Peroxidase (GPx) activity, Malondialdehyde (MDA) and Interleukin-1 Beta (IL-1β) of Obese Diabetic Rats.

Parameters / Groups	GPx	MDA	IL-1β
	U/ml	ng/ml	pg/ml
G1: -Ve Control	166.58±5.96^{a}	60.22±2.83^{e}	235.78±1.69^{e}
G2: +Ve Control (obese diabetic rats)	92.26±6.55^{e}	427.08±3.85^{a}	414.57±3.59^{a}
G3: 2.5% DBP	97.46±9.40^{d}	414.93±4.11^{a}	389.04±2.02^{b}
G4: 5% DBP	99.06±6.41^{d}	387.62±2.44^{b}	373.15±1.48^{c}
G5: 7.5% DBP	117.21±10.62^{c}	358.74±1.27^{c}	252.45±1.80^{d}
G6: 10% DBP	122.87±8.69^{b}	336.51±3.96^{d}	244.10±1.38^{de}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at P≤0.05.
*** DBP =** Dried Broccoli Powder.

Table 6: Effect of Broccoli on Serum Liver Functions of Obese Diabetic Rats.

Parameters / Groups	AST	ALT	ALP
	(µ/L)		
G1: -Ve Control	18.38±0.31^{d}	10.73±0.53^{e}	112.38±1.65^{e}
G2: +Ve Control (obese diabetic rats)	31.27±0.59^{a}	22.84±0.81^{a}	151.69±2.62^{a}
G3: 2.5% DBP	27.54±0.61^{ab}	21.05±0.51^{ab}	141.98±1.76^{b}
G4: 5% DBP	24.11±0.94^{bc}	18.17±0.38^{bc}	136.27±2.13^{bc}
G5: 7.5% DBP	23.15±0.51^{c}	17.85±0.28^{c}	133.43±1.54^{c}
G6: 10% DBP	21.62±0.50^{cd}	14.40±0.93^{d}	121.70±1.60^{d}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at P≤0.05.
*** DBP =** Dried Broccoli Powder.

Table 7: Effect of Broccoli on Serum Kidneys Functions of Obese Diabetic Rats.

Parameters / Groups	Urea	Creatinine	Uric Acid
	mg/dl		
G1: -Ve Control	42.52±0.78^{e}	0.70±0.01^{d}	3.50±0.17^{e}
G2: +Ve Control (obese diabetic rats)	63.65±0.43^{a}	2.38±0.17^{a}	7.47±0.22^{a}
G3: 2.5% DBP	58.98±1.04^{b}	2.05±0.08^{ab}	6.38±0.15^{b}
G4: 5% DBP	54.65±0.57^{c}	1.68±0.05^{b}	6.01±0.18^{bc}
G5: 7.5% DBP	51.25±0.47^{cd}	1.08±0.02^{c}	5.15±0.24^{cd}
G6: 10% DBP	49.51±0.51^{d}	0.81±0.05^{cd}	4.27±0.25^{de}

*Mean values are expressed as means ± SE.
*Mean values at the same column with the same superscript letters are not statistically significant at P≤0.05.
*** DBP =** Dried Broccoli Powder.

4. Conclusions

The present findings illustrate that broccoli is promising for the control of diabetes by reducing blood glucose and oxidative stress. This may be due to the presence of active components that have antioxidant activity.

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