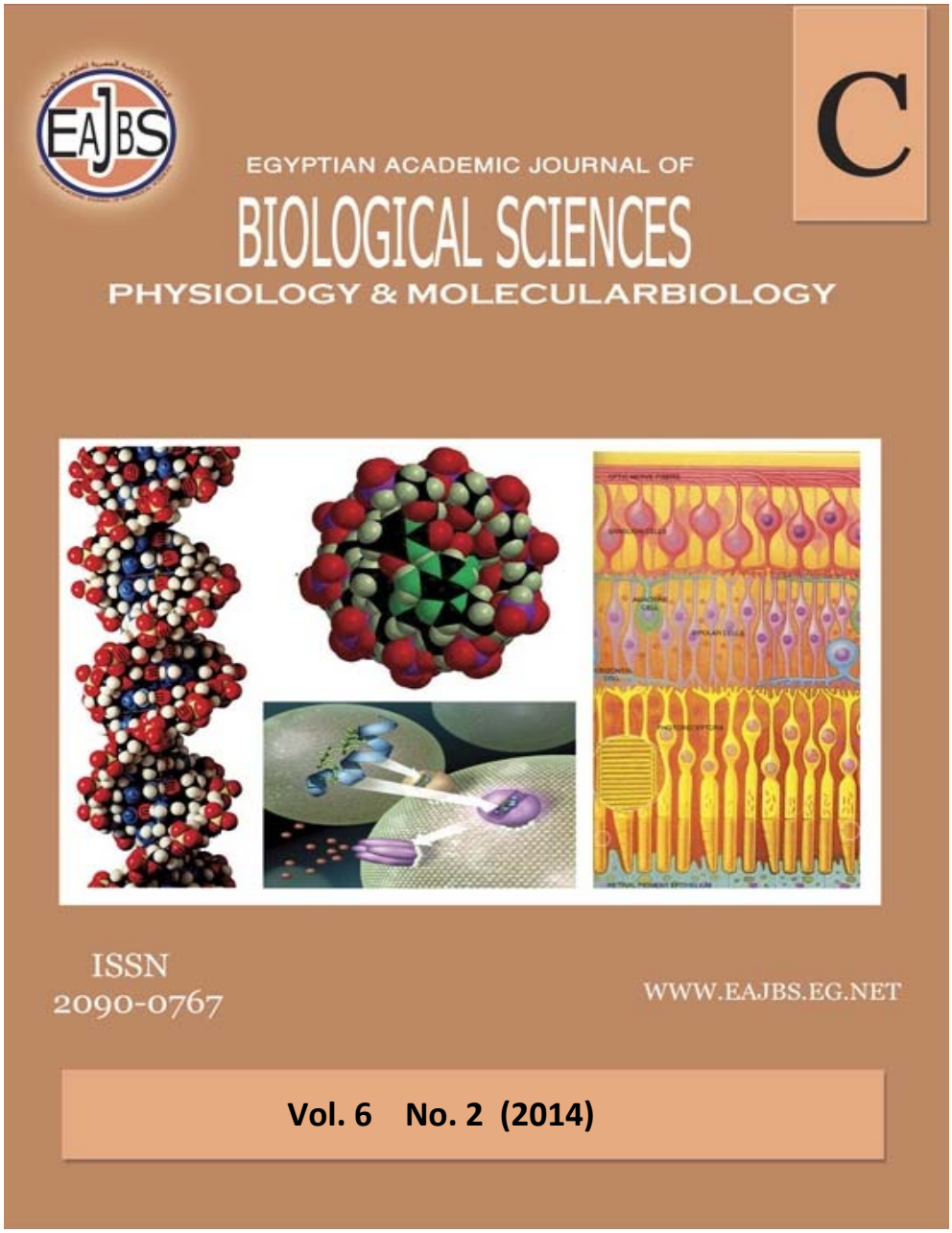

Egyptian Academic Journal of Biological Sciences is the official English language journal of the Egyptian Society for Biological Sciences ,Department of Entomology ,Faculty of Sciences Ain Shams University .

Physiology & molecular biology journal is one of the series issued twice by the Egyptian Academic Journal of Biological Sciences, and is devoted to publication of original papers that elucidate important biological, chemical, or physical mechanisms of broad physiological significance.

www.eajbs.eg.net

Egypt. Acad. J. Biolog. Sci., 6(2): 17 - 30 (2014)

Egyptian Academic Journal of Biological Sciences
C. Physiology & Molecular Biology
ISSN 2090-0767

www.eajbs.eg.net

Effect of White Tea Extract on Antioxidant Enzyme Activities of Streptozotocin –Induced Diabetic Rats

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ARTICLE INFO
Article History
Received: 1/4/2014
Accepted: 5/5/2014

Keywords:
Diabetes mellitus
Superoxide dismutase
Glutathione peroxidase
Catalase
Lipid peroxidation

ABSTRACT

Tea is the second most popular drink in the world after water, and many studies have highlighted the effects of drinking tea. Therefore, the aim of this study was to explore the effects of white tea extract on the antioxidant enzymes activity including superoxide dismutase (SOD), glutathione peroxidase (GSH-px), and catalase (CAT). A significant decrease was observed in serum and liver SOD, GSH-px, and CAT activities in the diabetic control rats compared with the nondiabetic control ones. However, a significant increase in SOD, GSH-px, and CAT activities (serum and liver) was observed in the diabetic groups treated with white tea extract or Glibenclamide as compared to the diabetic control group. In conclusion, the present findings indicated that white tea extract has an enhancing effect on the antioxidant enzyme activities (SOD, CAT, and GSH-px) in STZ-induced diabetic rats.

INTRODUCTION

Diabetes mellitus is a non-curable but controllable chronic metabolic disorder characterized by a loss of glucose homeostasis with disturbances of carbohydrate, fat, and protein metabolism due to defects in insulin secretion, insulin action, or both (American Diabetes Association 2012; DeFronzo 2004; WHO 1999). Lacking of sufficient insulin, leads body tissues, mainly; the liver and muscle tissues fail to capture and utilize glucose from the blood circulation. This resulted in the increase of blood-glucose levels, a state known as hyperglycemia (American Diabetes Association 2012). The restrictions of currently existing drugs have motivated researchers to explore alternative antidiabetic therapy. In particular, concern is given to plants and herbs used in traditional medicine as an antidiabetic treatment looking forward to discovering novel natural products that can be used as safe, cheap, and effective antidiabetic therapy (Kavishankar *et al.*, 2011).

Various medicinal plants such as *Allium sativa, Eugenia jambolana, Panax ginseng, Gymnema sylvestre, Momrodica charantia, Ocimum sanctum, Phyllanthus amarus,* and *Tinospora cordifolia* have been reported and confirmed for their hypoglycemic effect using experimental animal models of diabetes (Helmstadter and Schuster 2010; Khan *et al.,* 2012). In addition; hypoglycemic effect of some medicinal plants has been applied on diabetic patients in clinical studies (Ashraf *et al.,* 2011; Huyen *et al.,* 2010).

Tea is a product from the leaves of the plant *Camellia sinensis* (Family Theaceae). Tea production can be grouped into different types according to their degree of fermentation and processing: white, green, oolong, and black; all are widely used throughout the world as beverages and as folk medicines (Cabrera *et al.,* 2006). Tea is consumed by more than two-thirds of the world's population, and it is the most traditional beverage next to water. The increasing interest in the potential health benefits of tea, together with its popularity as a beverage, have encouraged many researchers to investigate the chemical constituents of tea (Gupta *et al.,* 2002; Sharma and Rao 2009), and their biological properties, such as antimutagenic (Bunkova *et al.,* 2005; Winn *et al.,* 2005), anticarcinogenic and antioxidant (Coyle *et al.,* 2008; Ferrazzano *et al.,* 2009; Karori *et al.,* 2007), antibacterial (Cho *et al.,* 2008; Prabhakar *et al.,* 2010), and antiallergic activities (Hassanain *et al.,* 2010). White tea is very similar to green tea, but it is exceptionally prepared only from leaves before the buds have been fully opened (the buds are still covered with fine white hair) and, hence, the name "white tea" (Sajilata *et al.,* 2008). White tea has been reported to have high polyphenolic contents and to exhibit antiseptic and antioxidant properties (Thring *et al.,* 2009, 2011). Various types of bioactive compounds are found in white tea such as polyphenol, caffeine, theogallin, gallic acid, theaflavin, flavonol glycosides, and catechins particularly epigallocathechins (EGC), epigallocatechins gallate (EGCG), epicatchin gallate (ECG), and epicatchin gallate (ECG) (Hilal and Engelhardt 2007; Rusak *et al.,* 2008). The concentrations of tea polyphenols, catechins, and related antioxidant activities are higher in white tea compared to green or black tea (Hilal and Engelhardt 2007; Venditti *et al.,* 2010). Kumar *et al.* (2012) showed that white tea is effective in fighting benzo (a) pyrene induced oxidative stress and DNA damage. Santana-Rios *et al.* (2001), found that there are greater antimutagenic properties in white tea. Recent investigation associated white teas with anticarcinogenic, immuneboosting, and antioxidative properties that may impact human health in a manner comparable to green teas (Unachukwu *et al.,* 2010). Therefore, the aim of this study was to explore the effect of white tea extract on antioxidant enzyme activities.

MATERIALS AND METHODS

Materials

All the chemicals and reagents used in this work were of the highest grade available. Kits for determination of the enzymatic activities of SOD and GSH-px were from Randox, UK. Catalase assay kit from BioVision, USA, and GSH from Biodiagnostic, Egypt.

Animals

Male white albino rats (*Rattus norvegius*) weighing 170–200g were obtained from the Experimental Animal Unit, College of Science, King Khalid University, Saudi Arabia and were used in this study. All rats received food and water *ad libitum* and were kept in a room with the temperature regulated to 22 ± 1°C. The experiment was approved by

the Animal Ethical Committee, College of Science, King Khalid University.

Methods

Diabetes Induction

In this study, diabetes was induced by a single intraperitoneal (i.p.) injection of streptozotocin (STZ) at a dose of 55mg/kg body weight, freshly dissolved in 0.1M sodium citrate buffer (pH 4.5) within 10 min after preparation to 16h fasted rats (Aslan *et al.,* 2007; Aslan *et al.,* 2010; Renno *et al.,* 2008a). Control rats received an equivalent amount of the sodium citrate buffer (pH 4.5). For the i.p. injection of STZ, the rat was held in one hand in dorsal position; the injection site was swabbed using alcohol swaps, and the designated amount of STZ was injected into the caudal abdominal cavity using a sterile needle. Since STZ can produce fatal hypoglycemia as a result of pancreatic release of insulin, the rats were treated with 20% glucose solution after 1h. To overcome the hypoglycemia, which occurred during the first 24h following the STZ administration, 5% glucose solution bottle was given to the diabetic rats (Jayaraman *et al.,* 2009). Diabetes was confirmed by determination of fasting blood glucose after 48h of streptozotocin administration, whole blood samples were taken from the tail vein of rats, and the blood-glucose levels were estimated in rats following overnight fasting by Roche Accu check active glucometer & Accu check active strips (Agrawal *et al.,* 2010). Rats with a blood glucose ranging above 300mg/dl were considered diabetic and used in the experiment.

Preparation of White Tea Extract

Commercially available white tea bags were prepared in 2% (w/v) as follows: 20 g white tea in bags was prepared freshly by steeping in 1L of boiled distilled water in a covered container for 5 minutes. The tea was cooled to room temperature and filtered before administration to the rats in clean drinking bottles (Renno *et al.,* 2008a; Renno *et al.,* 2008b; Tas *et al.,* 2005). Each aqueous solution (water/tea) was given to the animals for four weeks.

Preparation of Glibenclamide Drug Solution

Glibenclamide belongs to second generation sulfonylureas. In this study, it was taken as the standard drug. Glibenclamide was obtained from Assir Central hospital, Abha, Saudi Arabia, in the form of a 5mg tablet. The tablets were finely powered and suspended in distilled water at a concentration of 5mg/ml (Kurgaliuk and Tkachenko 2006). Glibenclamide was given daily to diabetic standard control group (as a 600µg/kg body weight) using an intragastric tube (Nagappa *et al.,* 2003).

Experimental Design

Thirty rats (18 diabetic surviving rats, twelve normal rats) weighing 170-200g were used in this study. The rats were divided into five groups of six rats each as shown in Table 1. Animal experiments were approved by the animal research ethical committee, college of science, King Khalid University. The treatment period with aqueous tea extract was four weeks.

Table 1: Groups used in this study.

Group	Treatment
Group I, Normal Control	Normal rats received water
Group II, Normal Control + white tea	Normal rats received white tea extract
Group III, Diabetic Control	Diabetic rats received tap water
Group IV, Diabetic +white tea	Diabetic rats received white tea extract
Group V, Diabetic + Glibenclamide treatment (D + Glib)	Diabetic rats treated with the anti-diabetic drug, Glibenclamide (600µg/kg body weight)

Preparation of Serum and Homogenate

After the final oral dosing (end of the experimental period), rats were fasted overnight (~16h) by removing all chow from the cages, while water remained available *ad libitum.* Diabetic and the corresponding control animals were sacrificed by decapitation under light ether anesthesia. The procedure described by Nafiu *et al.* (2011) was used in preparation of serum. Briefly, the rats were anaesthetized in either vapor, when they became unconscious, their neck areas were quickly cleaned of fur and skin to expose the jugular veins, then the vein was cut with sterile scalpel and made to bleed into a clean, dry centrifuge tube and allowed to clot for 10min before being centrifuged at 2,000 rpm for 10min at 4°C in a refrigerated centrifuge. The sera was thereafter aspirated into clean, dry, sample eppendorf tubes using Pasteur pipette and stored at -80°C until it was used for the estimation of the biochemical parameters.

For Biochemical analysis, the extract was prepared by homogenizing 0.5g of the tissue in 5.0ml of PBS using an electric homogenizer. The homogenate was filtered through cheese cloth, and the filtrate was centrifuged for 15min at 6000rpm using a refrigerated centrifuge to remove nuclear fraction. The resultant supernatant was used for the estimation of antioxidant enzymes: glutathione peroxidase, superoxide dismutase, and catalase. The protein contents in the cytosolic fraction were determined by the Bradford method (1976) with bovine serum albumin as a standard.

Biochemical and Enzymes Assays

Lipid Peroxidation. Lipid peroxidation is a chain of reactions that involves the oxidation of polyunsaturated fatty acids in membranes by free radicals and considered as a marker of oxidative cell damage (Halliwell and Gutteridge 1984). The direct measurement of oxidative stress in the laboratory is difficult because the free radicals have a very short half-life, as an alternative, products of the oxidative process are measured. When a fatty acid is peroxidized it is broken down into aldehydes, which are excreted. Aldehydes such as thiobarbituric acid reacting substances (TBARS) have been widely accepted as a general marker of free radical production (Clarkson 1995). The most commonly measured TBARS is malondialdehyde (MDA).

The pink colored product generated as a result of the reaction between malondialdehyde (MDA) and thiobarbituric acid (TBA) reagent under acidic conditions were assessed in the tissues by the method of Ohkawa *et al.* (1979) and Yagi (1998) at 532nm. In brief, the tissue supernatant (0.5ml) or 0.5ml (blank) was added to test tubes containing 1.0ml of thiobarbituric acid reagent (mixture of 0.375g TBA, 15g TCA and 0.25ml HCL). The reaction mixture was incubated in boiling-water bath for 30min. After cooling to room temperature for 10min, the tubes were centrifuged at 4000rpm for 10min. The supernatant layer was used for spectrophotometric measurement at 532nm. The concentration of MDA is calculated using an extinction coefficient of MDA-TBA complex, which is 1.56 $x10^5$ $M^{-1}cm^{-1}$ and the results was expressed as nmole MDA /mg protein.

Determination of Glutathione (GSH) Concentration

Glutathione (GSH) an endogenous antioxidant, was detected spectrophotometrically as described by Beutler (1963) using glutathione reduced colorimetric method (Biodiagnostic), briefly, 100µl of the sample and 100µl of 25% TCA were mixed and allowed to at room temperature for 10min then the mixture was subjected to centrifugation at 3000rpm for 15min to settle the

precipitate. 500µl of the supernatant was taken in a test tube containing the 1ml of 0.6mM 5,5'-dithiobis (2-nitrobenzoic acid) [DTNB] and 100µl of 0.2mM sodium phosphate buffer (pH 7.4). The yellow colored complex obtained was measured at 405nm against the reagent blank which contained 100µL of 25% TCA in place of the supernatant).

Determination of Superoxide Dismutase (SOD) Activity

SOD activity was determined in the sample according to the method of Wooliams *et al.* (1983) using Randox superoxide dismutase kit. The sample (30µl) was mixed with 1000µl mixed substrate [Xanthine (0.05mmol/l), I.N.T. (0.025 mmol/l), CAPS (50mmol/l), and EDTA (0.94mmo/l)], followed by the addition of 150µl of xanthine oxidase. Absorbance was read at 505nm using a spectrophotometer. The initial absorbance was read 30 seconds after addition of xanthine oxidase. The final absorbance was read three minutes after the first reading. A change in absorbance per minute was calculated, and the percentage inhibition determined by comparing the reaction to the uninhibited reaction (xanthine oxidase added to the mixed substrate). A standard curve was plotted for each standard against the percentage of inhibition, which was used to determine the superoxide dismutase activity (U/ml).

Determination of Glutathione Peroxidase (GSH-px) Activity

GSH-px activity in the sample was measured according to the Paglia and Valentine's method (1967) using Randox GSH-Px kit. This method based on the oxidation of glutathione by cumene hydroperoxide. In the presence of glutathione reductase and NADPH, the oxidized glutathione is immediately converted to the reduced form with a concomitant oxidation of NADPH to NADP+. The decrease in absorbance at 340nm was measured. One unit of GSH-px is equivalent to the amount of enzymes necessary to reduce 1µM of NADPH per minute at 37°C under specified conditions. The activity of GSH-px was measured at 340nm by measuring the decrease of NADPH absorbance using an extension coefficient of $6.22mM^{-1}cm^{-1}$. The assay contained 20µl of the sample; 1ml R1 [reduced glutathione (4mmol/l), NADPH (0.34mmol/l), glutathione reductase (≥0.05U/ml), phosphate buffer (0.05mol/l; pH 7.2), EDTA (4.3mmol/l)]. The reaction was started by adding 40µl R2 [cumene hydroperoxide (0.18mmol/l)]. The sample, R1 and R2 were mixed and the initial absorbance of the sample and blank reagent was measured spectrophotometrically at 340nm after one minute and start timer simultaneously. Read again after 1 and 2 minutes.

Determination of Catalase activity

Catalase was measured using the Catalase Activity Assay Kit (BioVision). The assay involves two steps: catalase first reacts with H_2O_2 to produce water and oxygen. The remaining (unconverted) H_2O_2 reacts with OxiRed™, probe to produce a product, which can be measured by a colorimetric method. The sample and positive control were performed as follows: 10µl serum, 5µl of tissue (or positive control solution) samples or were added individually into each well, and the volume was adjusted to total 78µl with assay buffer. To prepare the sample High Control (HC), 1µl of the sample was added into separate well followed by adjusting the total volume to 78µl with assay buffer. 10µl of stop solution was added only into the sample HC well, followed by mixing and incubation at 25°C for 5min to completely inhibit the catalase activity in samples as High Control. The H_2O_2 standard curve was prepared as follows: 20mM H_2O_2, 5µl of 0.88M H_2O_2 was added to 215µl dH_2O, then 50µl of the 20mM H_2O_2 was added to 950µl dH_2O to generate 1mM H_2O_2. H_2O_2 standard was

prepared by the addition of 0, 2, 4, 6, 8µl of 1mM H_2O_2 solution into 96-well plate to create 0, 2, 4, 6, 8nmol/well. The final volume in each well was adjusted to 90µl with assay buffer. The reaction was stopped by the addition of 10µl stop solution into each well. The catalase reaction was started by the addition of 12µl of fresh 1mM H_2O_2 into each well of both samples and sample HC, followed by incubation at 25°C for 30min, then 10µl of the stop solution was added into each sample well only (Note: High Control and standard curve wells already contain stop solution). 50µl of the developer mix (46µl Assay Buffer, 2µl OxiRed™ Probe, 2µl HRP solution) was added to each test sample, controls, and standards. The wells were mixed well and incubated at 25°C for 10min, then 10µl of the stop solution was added followed by mixing and reading the absorbance at 570nm in a plate reader.

Statistical Analysis

The data for various biochemical parameters were statistically analyzed using Statistical Package for the Social Sciences (SPSS) software (SPSS 2007). Comparison between group means was done using Analysis of Variance (ANOVA) and Post hoc least significant differences (LSD). Results were presented as means ± S.E. $P<0.05$ were considered as statistically significant.

RESULTS

Induction of Diabetes

Table 2 shows that all rats injected with STZ developed severe diabetes as indicated by significant increase ($P<0.05$) in blood-glucose concentrations range (425-460nmg/dl) which represents approximately threefold as compared with nondiabetic control rats. Before treatment started, the rats had comparable levels of blood-glucose (113.4 ± 6.2 mg/dl).

Table 2: Blood glucose concentrations of nondiabetic and diabetic rats.

Groups	Glucose concentration (mg/dl)
Non diabetic	113.4±6.2
Diabetic	425.8 ±37.2[a]

[a] represents the significance of the difference compared with the nondiabetic rats

Effect of Extract on Lipid Peroxidation

Figure 1 showed a significant increase ($P<0.05$) in the liver MDA level in the diabetic control rats (6.07 ± 0.459 nmol/mg protein) compared with the nondiabetic control rats (3.99 ± 0.263 nmol/mg protein). However, results of this study showed that white tea extract could significantly decrease ($P<0.05$) the formation of MDA in STZ-treated rats to 4.53 ± 0.25 nmol/mg protein. At the same time, the effect of Glibenclamide on MDA levels in STZ-treated rats was found to be reduced to 3.79 ± 0.19 nmol/mg protein (Figure 1).

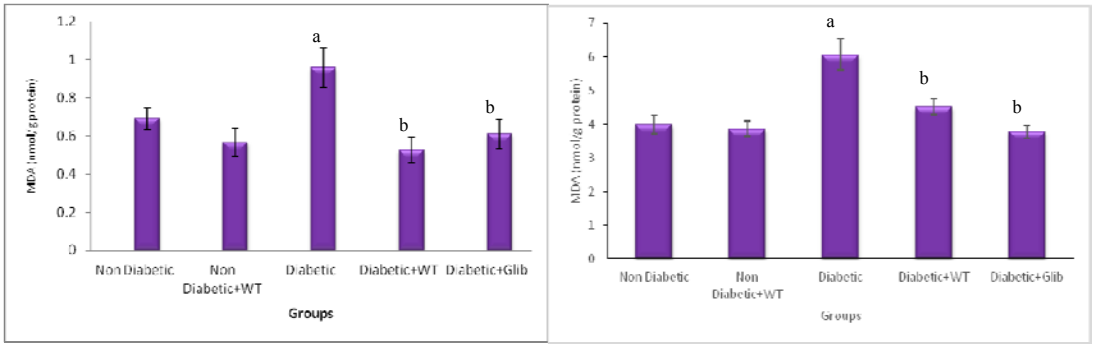

Fig. 1: Serum and liver Malondialdehyde concentration in different groups at the end of the four week experimental period.
Data are shown as mean ± SE.
[a] represents the significance of the difference compared with nondiabetic control rats.
[b] represents the significance of the difference compared with diabetic control rats.

Effect of White Tea Extract on Glutathione Levels (GSH)

The present study showed a significant decrease (P<0.05) in the serum and liver GSH of the diabetic rats compared with the nondiabetic rats due to effect of STZ treatment, which enhances the level of lipid peroxidation, whereas treatment with white tea significantly increased (P<0.05) the GSH levels (Figure 2). This suggests that white tea extract may reduce ROS. Therefore, decrease the oxidative damage. No significant difference was found when the on the level of serum or liver GSH of diabetic rats treated with white tea or Glibenclamide.

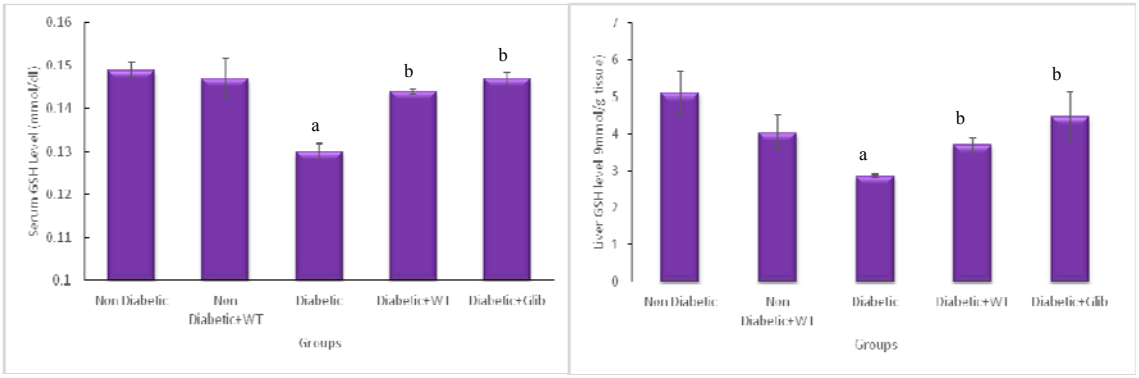

Fig. 2: Serum and liver glutathione concentration in different groups at the end of the four week experimental period.
Data are shown as mean ± SE.
[a] represents the significance of the difference compared with nondiabetic control rats.
[b] represents the significance of the difference compared with diabetic control rats.

Effect of White Tea Extract on the Activities Superoxide Dismutase

The rat serum SOD activity was significantly (*P<0.05*) reduced in the diabetic control group in comparison to the nondiabetic control rats (Figure 3). Under the experimental conditions of this study, white tea extract improved the antioxidant status in the diabetic rats by significantly increasing (*P<0.05*) SOD activity after four weeks of treatment in both serum and liver tissue (84.35 ± 4.4, 5.728 ± 0.62U/ml, respectively) in comparison to the diabetic control rats (70.77 ± 3.7, 4.55 ± 0.35U/ml, respectively). The same trend was observed in the diabetic rats treated with Glibenclamide (Figure 3).

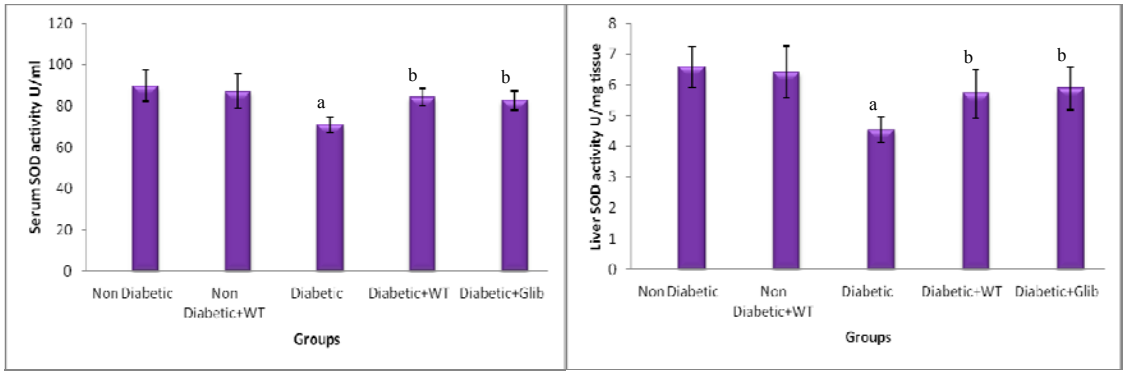

Fig. 3: Serum superoxide dismutase activity in different groups at the end of the four week experimental period.
Data are shown as mean ± SE.
[a] represents the significance of the difference compared with nondiabetic control rats.
[b] represents the significance of the difference compared with diabetic control rats.

Effect of White Tea Extract on the Glutathione Peroxidase

SZT treatment caused significant decreased (P<0.05) in the level of glutathione peroxidase (GSH-px) activity in serum and liver tissue when compared to with the control group (serum: 0.124 ± 0.03 and 0.273 ± 0.02; liver: 3.920 ± 0.66, and 7.99 ± 0.58µmol/min/g tissue, respectively, Figure 4). The treatment of white tea extract resulted in significant increase (P<0.05) when compared to the diabetic control rats.

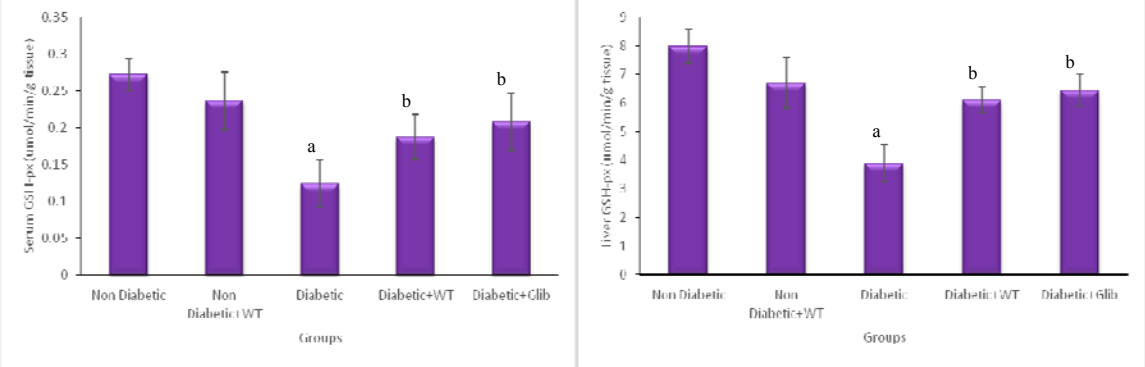

Fig. 4: Serum and liver glutathione peroxidase activity in different groups at the end of the four week experimental period.
Data are shown as mean ± SE.
[a] represents the significance of the difference compared with nondiabetic control rats.
[b] represents the significance of the difference compared with diabetic control rats.

Effect of White Tea Extract on Catalase

A significant decrease (P<0.05) was observed in serum and liver catalase (CAT) activity in the diabetic control rats by 0.690 ± 0.046 and 4.697 ± 0.254, respectively compared with the nondiabetic control rats (Figure 5). Furthermore, significant increase (P<0.05) in CAT activity (serum and liver) was observed in the diabetic groups treated with white tea extract or Glibenclamide as compared to the diabetic control group (Figure 5).

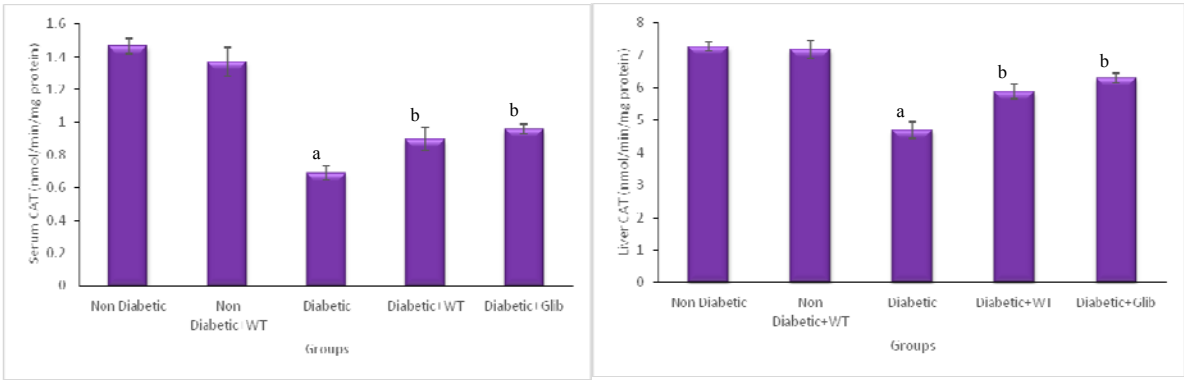

Fig. 5: Serum and liver catalase activity in different groups at the end of the four week experimental period.
Data are shown as mean ± SE.
[a] represents the significance of the difference compared with nondiabetic control rats.
[b] represents the significance of the difference compared with diabetic control rats.

DISCUSSION

As diabetes and its complications are associated with free radical mediated cellular injury (Kaleem *et al.,* 2006), an aqueous extract of white tea was administered to diabetic rats to assess their antioxidant potential. Diabetes is well-known to be associated to an unbalanced production of ROS, such as hydroxyl radicals (HO), superoxide anions (O_2), and H_2O_2. Autoxidation of glucose arises during hyperglycemia, may cause disruption of cellular function and oxidative damage of cell membranes due to increased level of free radicals (Evelson *et al.,* 2005; Hussein 2008). Therefore, as a safeguard against the accumulation of these free radicals, intracellular enzymatic antioxidant activities exist. Free radicals generated during metabolism can enter into reactions that, when uncontrolled, can affect the cell components such as proteins, lipids, carbohydrates, and DNA of healthy cells leading to clinical manifestations (Mano *et al.,* 2000; Nunez *et al.,* 2013). Davi *et al.* (2005) showed that hyperglycemia inactivate antioxidant enzymes like SOD, CAT, and GSH-px and stimulate oxidative stress, which causes lipid peroxidation. However, oxidative stress can be prevented by antioxidants and over expression of antioxidant enzymes (Kaneto *et al.,* 2007).

In this study, the effect of white tea on biomarkers of oxidative stress in serum and liver homogenate of diabetic rats was investigated. The localization of radical formation resulting in lipid peroxidation, measured as Malondialdehyde (MDA). An elevation in the levels of MDA in the diabetic rats indicates the enhanced lipid peroxidation which leads to tissue damage and also free radical generation above the cellular radicals scavenging capacity, which means the failure of the antioxidant defense mechanism (Maxwell *et al.,* 1997). In the present study, a significant decrease in MDA levels in the diabetic rats treated with white tea extract was observed as compared to diabetic control where the levels were significantly high. This indicates that white tea may inhibit lipid peroxidation and thereby oxidative damage to tissues and organs in diabetes. The hepatoprotection of white tea may be due to its antioxidant activity.

Glutathione (GSH) is one of the most abundant tripeptide, non-enzymatic biological antioxidant present in the liver. GSH is helpful for the removal of free radicals such as H_2O_2 and superoxide radicals, detoxification of foreign chemicals and biotransformation of drugs (Cacciatore *et al.,* 2010; Wu *et al.,* 2004). In the present study, treatment with white tea extract significantly increased the glutathione content in blood when compared to diabetic control rats where the levels were significantly decreased. This increased GSH content in blood of the rats treated with white tea extract may be one of the reasons responsible for the inhibition of lipid peroxidation.

Superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GSH-px) are the three major scavenging enzymes that remove the toxic free radicals *in vivo*. The decreased activity of antioxidant (SOD, CAT, and GSH-px) enzymes along with elevated lipid peroxide levels in diabetic rats was found in the liver during the diabetic state, which could probably be associated with oxidative stress and/or decreased antioxidant defense potential; these results are consistent with the results of previous studies (Derosa *et al.,* 2013; Evelson *et al.,* 2005; Gallou *et al.,* 1993; Hussein 2008; Sun *et al.,* 2013). Kaleem *et al.* (2013) declared that the decreased in the activities of SOD and catalase in the liver during diabetic status may be due to the accumulation of superoxide

radicals and hydrogen peroxides. SOD has been postulated as one of the most important enzymes in the enzymatic antioxidant defense system which catalyses the dismutation of superoxide radicals to produce H_2O_2 and molecular oxygen (Mc Crod *et al.,* 1976). The observed decrease in the diabetic rats SOD activity could result from inactivation by H_2O_2 or by glycation of enzymes (Sozmen *et al.,* 2001).

Administration of white tea extract significantly increased the activities of SOD, CAT, and GSH-px in serum and liver thus indicating a protective effect. This means that the extract may reduce reactive oxygen-free radicals and improve the activities of antioxidant enzymes associated with diabetes through the inhibition of lipid peroxidation. The protective effect of white tea may act by either directly scavenging the reactive oxygen metabolites, due to the presence of various antioxidant compounds, or by increasing the synthesis of antioxidant molecules (Hamden *et al.,* 2008). These results are in agreement with other studies on green tea supplementation in diabetic rats and other hepatic dysfunction, which showed an increase in the levels of antioxidant enzymes, which may be associated with its components that have been scavenging free radical properties. (Hamden *et al.,* 2008; Mustata *et al.,* 2005). These studies showed that flavonoids existing in tea extract to enhance the expression of intracellular antioxidants such as SOD, catalase and GSH-px by retaining their activities higher compared to the control treated rats (Hamden *et al.,* 2008).

ACKNOWLEDGMENT

The authors wish to thank King Abdulaziz City for Science and Technology, Saudi Arabia for financial support.

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ARABIC SUMMARY

تأثير مستخلص الشاي الأبيض على نشاط الإنزيمات المضادة للأكسدة في الجرذان الطبيعية والمصابة بمرض السكري

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يعتبر الشاي في وقتنا الحاضر ثاني أشهر مشروب في العالم بعد الماء، وقد سلطت العديد من الدراسات الضوء على التأثيرات الإيجابية لمشروب الشاي. لذا فقد هدفت هذه الدراسة إلى بيان تأثير المستخلص المائي للشاي الأبيض على مرض السكري المستحدث بمركب الستربتوزوتوسين على بعض الإنزيمات المضادة للأكسدة وذلك بقياس نشاط انزيمات (SOD) و(GSH-px) و(CAT)، في كل من المصل و الكبد. أجريت التجارب على 30 من ذكور الجرذان البيضاء تراوحت أوزانها ما بين 170-200غم. وقد تم استحداث مرض السكري بحقن الجرذان عبر التجويف البريتوني بجرعة مفردة من مركب الستربتوزوتوسين بتركيز 55ملغم/كغم من وزن الجرذ. وقد قسمت الجرذان إلى خمسة مجموعات كل مجموعة مكونة من ستة جرذان كما يلي: المجموعة الأولى المجموعة الضابطة السالبة غير المصابة بالسكري؛ المجموعة الثانية غير مصابة بمرض السكري ومعاملة بالشاي الأبيض؛ المجموعة الثالثة المجموعة الضابطة الموجبة المصابة بمرض السكري؛ المجموعة الرابعة مصابة بمرض السكري ومعاملة بالشاي الأبيض والمجموعة الخامسة مصابة بمرض السكري ومعاملة بعقار الجلايبنكلاميد (العقار المتداول تجارياً لخفض السكر في الدم). وقد طبق نظام غذائي موحد على جميع المجموعات بحيث يكون الشراب الوحيد إما ماء عادي أو مستخلص الشاي الأبيض بنسبة 2% طول مدة الدراسة والتي استمرت لمدة أربعة أسابيع. أوضحت نتائج هذه الدراسة أن هناك ارتفاعاً ذو دلالة معنوية في نشاط كل الإنزيمات المضادة للأكسدة في مصل كل الجرذان المصابة بمرض السكري والمعاملة بالشاي الأبيض مقارنة بالجردان المصابة بمرض السكري.