Effect of *Cichorium endivia* Leaves on Some Biochemical Parameters in Streptozotocin-Induced Diabetic Rats

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Abstract: The present study was carried out to examine the effect of aqueous suspension of *Cichorium endivia* leaves powder on some biochemical parameters in streptozotocin-induced diabetic rats. Adult male albino rats were randomly divided into 5 groups, (1) control group which was administered distilled water, (2) *Cichorium endivia* group in which rats received aqueous suspension of *C. endivia* leaves powder, 500 mg/kg b. wt., (3) diabetic group in which diabetic rats were administered distilled water, (4) diabetic–*Cichorium endivia* group in which diabetic rats were treated with aqueous suspension of *C. endivia* leaves powder, 500 mg/kg b. wt., and (5) diabetic–glibenclamide group in which diabetic rats were treated with glibenclamide drug, 600 mg/kg b. wt. All groups were induced by intragastric administration for six weeks. The *C. endivia* leaves powder or the glibenclamide drug produced significant hepatoprotective effects by decreasing the activities of serum aminotransferases (AST and ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and liver malondialdehyde (MDA) level as well as liver superoxide dismutase (SOD) and catalase (CAT) activities, and increasing the liver glutathione peroxidase (GPx) and glutathione-S-transferase (GST) activities and reduced glutathione (GSH) level.

In conclusion, the results obtained clearly indicate the role of oxidative stress in induction of diabetes and suggest protective and/or ameliorative effects of *C. endivia* leaves powder aqueous suspension, similar to the diabetic drug (glibenclamide) effects in this animal model.

Key words: *Cichorium endivia*- Liver- Streptozotocin- Biochemical parameters- Diabetes- Antioxidant enzymes

INTRODUCTION

Diabetes is a major degenerative disease in the world today (Ogbonnia et al., 2008). It is a multifactorial disease which is characterized by hyperglycemia (Ugochukwu et al., 2003), lipoprotein abnormalities (Scopolla et al., 2001), raised basal metabolic rate (Owu et al., 2006), defect in reactive oxygen species scavenging enzymes (Kesavulu et al., 2000) and high oxidative stress induced damage to pancreatic β-cells (Nayeemunnisa, 2009). Streptozotocin (STZ) was recognized as a toxic agent for β-cells of the islets of Langerhans (Agarwal, 1980), and has since then been widely used for the induction of diabetes mellitus in experimental animals (Kim et al., 2003). Damasceno et al. (2002) reported that STZ produced oxidative stress and depletion of antioxidant systems in both blood and tissue particularly, liver. The concentration of the reactive oxygen species are modulated by antioxidant enzymes-glutathione peroxidase (GPx), glutathione-S-transferase (GST), superoxide dismutase (SOD), catalase (CAT) and non-enzymatic scavengers like reduced glutathione (GSH) (Saxena et al., 1993).

Management of diabetes mellitus without any side effects is still a challenge to the medical system. There is an increasing demand by patients to use natural products with antidiabetic activity, because insulin and oral hypoglycemic drugs have undesirable side effects (Kameswara and Appa, 2001). Medicinal plants are a good source of natural antioxidants believed to exert their effects by reducing the formation of the final active metabolite of the drug-induced systems or by scavenging the reactive molecular species to prevent their reaching a target site (Kaleem et al., 2005). It has been documented that several medicinal plants show them fromhypoglycemic effects associated with a significant alteration in the activity of liver hexokinase (Bopanna et al., 1997) and glucokinase (Kumari et al., 1995). In addition, Mansour et al. (2002) and Eliza et al. (2009) demonstrated that the administration of several herb extracts could restore the changes in the activities of serum enzymes, like transaminases (AST & ALT) as well as alkaline phosphatase (ALP) and lactate dehydrogenase (LDH) of diabetic rats.

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Cichorium endivia, a member of the sunflower family Asteraceae, is a typical Mediterranean plant indigenous to Europe, Western Asia and North America (Fernald, 1950). It is very valuable nutritionally, with a high content of dietary fibres, potassium and vitamin C (Kopeck, 1998). The antibacterial, antimalarial, cytotoxic, anti-diabetic, no-mutagenic and other activities of chicory were evaluated previously (Petrovic et al., 2004). Earlier investigations have reported that the ethanol extract of Cichorium intybus has anti-diabetic and hypolipidemic activities in STZ-induced diabetic rats (Pushparaj et al., 2007). Similarly, Upur et al. (2009) mentioned that Cichorium glandulosum extract reduced serum AST, ALT and ALP activities in CCl4 and galactosamine administration, which induced acute hepatotoxicity in mice, these results suggested that C. glandulosum is a potent hepatoprotective agent that could protect liver against the acute injury and this ability might be attributed to its antioxidant potential. Therefore, the present study aimed to examine the effect of C. endivia leaves powder (aqueous suspension) or the glibenclamide drug on some biochemical parameters of STZ-induced diabetic rats.

MATERIALS AND METHODS

Animals:

Adult male albino rats (Rattus norvegicus) weighing 105-150 g were used as experimental animals in this study. These animals were grouped and housed in polyacrylic cages (five animals per cage). The rats were provided with standard diet and water ad libitum. They were acclimatized under laboratory conditions for one week before experimentation.

Chemicals and Drug:

Streptozotocin (STZ) was purchased from Sigma – Aldrich (St Louis, MO, USA). The glibenclamide drug (commercial name Daonil) was purchased from Aventis Pharma S.A.E., Germany. This drug was widely used in Egypt as diabetic treatment and control.

Plant Material:

Cichorium endivia is aerb belonging to the Asteraceae family. The plant leaves were collected from fields in Giza Province, Egypt. The plant leaves were identified and authenticated by the Herbarium of Botany Department, Faculty of Science, Cairo University. The leaves were dried in an incubator and grounded to a fine powder with a grinder. The C. endivia leaves powder was stored in dry place until used.

Experimental Design:

Animals were randomly divided into five groups (7 rats/group), the 1st group served as normal control group (N.C.); rats of this group were administered daily 1 ml distilled water orally by gastric gavage. The 2nd group: normal Cichorium endivia group (N.Ch.); rats were orally administered with aqueous suspension of C. endivia leaves powder (500 mg/kg b. wt.) according to Sadeghi et al. (2008). The 3rd group: diabetic control group (D.C.); rats were rendered diabetic by a single intraperitoneal (I.P.) injection of STZ (50 mg/kg b. wt.) according to Kanter et al. (2003), in freshly prepared sodium citrate buffer (0.1 M, pH 4.5) after an overnight fast. After 48 hours of STZ injection, blood glucose level of each rat was determined. Rats with blood glucose range from 200-300 mg/dl were considered diabetic and included in the study. Rats of this group were administered daily 1 ml distilled water orally after 48 hours of STZ injection. The 4th group: diabetic–Cichorium endivia group (D.Ch.); diabetic rats were administered daily aqueous suspension of C. endivia leaves powder (500 mg/kg b. wt.) orally. The 5th group: diabetic–glibenclamide group (D.G.); diabetic animals were administered orally glibenclamide drug (600 mg/kg b. wt.) according to Chandramohan et al. (2009). At the end of the experimental period (six weeks), the rats were sacrificed; blood and liver samples were collected for different biochemical analyses.

Serum Preparation:

Blood samples were collected in centrifuge tubes and centrifuged at 3000 rpm for 20 min. Serum was stored at -20°C until used for biochemical assays. The appropriate kits (Biodiagnostic kits) were used for determination of the activities of serum aminotransferases AST & ALT according to Reitman and Frankel (1957), alkaline phosphatase (ALP) (Belfield and Goldberg, 1971) and lactate dehydrogenase (LDH) (Vassault et al., 1986).
Liver Tissue Preparation:
Liver was homogenized (10% w/v) in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 3000 rpm for 20 min. at 4°C. The resultant supernatant was used for determination of the activities of liver catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione-S-transferase (GST) according to the methods described by Aebi (1984), Nishikimi et al. (1972), Paglia and Valentine (1967) and Habig et al. (1974), respectively, as well as the levels of reduced glutathione (GSH) (Beutler et al., 1963) and malondialdehyde (MDA) (Ohkawa et al., 1979) using Biodiagnostic kits.

Statistical Analysis:
Values were expressed as mean ± SE. To evaluate differences between the groups studied, one way analysis of variance (ANOVA) with LSD post hoc test was used to compare the group means and P<0.05 was considered statistically significant. SPSS, for Windows (version 17.0) was used for the statistical analysis.

RESULTS AND DISCUSSION

Amelioration of Liver Functions in C. endivia treated Diabetic Rats:
The activities of serum AST, ALT, ALP, and LDH in the N.C., N.Ch., D.C., D.Ch. and D.G. are presented in Table 1 and Fig. 1. The results showed that the administration of aqueous suspension of C. endivia leaves powder to normal rats caused significant decrease (P<0.05) in serum AST, ALT, ALP, and LDH activities, as compared to the control group. On the other hand, the activities of these enzymes increased significantly (P<0.05) in serum of STZ-induced diabetic rats, as compared to the control group. Treatment of diabetic rats orally with aqueous suspension of C. endivia leaves powder caused significant decrease (P<0.05), of -8.10%, -22.88%, -9.45% and -12.77% in serum AST, ALT, ALP, and LDH activities, respectively, as well as treatment of diabetic rats with glibenclamide drug also caused significant decrease (P<0.05), of -8.12%, -19.89%, -15.59% and -13.28% in these serum enzymes, activities respectively, as compared to the diabetic group. It seems that C. endivia leaves powder and glibenclamide drug are useful in improvement the alteration recorded in such serum enzymes activities in the diabetic rats (Table 1 & Fig. 1).

Table 1: Effect of treatment with aqueous suspension of C. endivia leaves powder or glibenclamide drug for 6 weeks on serum AST, ALT, ALP and LDH activities of diabetic rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>N.C.</th>
<th>N.Ch.</th>
<th>D.C.</th>
<th>D.Ch.</th>
<th>D.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST (U/ml)</td>
<td>64.09 ± 0.79</td>
<td>60.70 ± 1.06</td>
<td>74.30 ± 1.02</td>
<td>68.28 ± 0.81</td>
<td>68.27 ± 1.77</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>15.93</td>
<td>-8.10</td>
<td>30.86</td>
<td>54.90</td>
<td>16.76</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>52.37 ± 1.40</td>
<td>44.26 ± 1.01</td>
<td>68.53 ± 2.60</td>
<td>52.85 ± 0.92</td>
<td>54.90 ± 1.67</td>
</tr>
<tr>
<td>ALT (U/ml)</td>
<td>52.37 ± 1.40</td>
<td>44.26 ± 1.01</td>
<td>68.53 ± 2.60</td>
<td>52.85 ± 0.92</td>
<td>54.90 ± 1.67</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>30.86</td>
<td>-22.88</td>
<td>-19.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>52.37 ± 1.40</td>
<td>44.26 ± 1.01</td>
<td>68.53 ± 2.60</td>
<td>52.85 ± 0.92</td>
<td>54.90 ± 1.67</td>
</tr>
<tr>
<td>ALP (IU/L)</td>
<td>86.58 ± 2.56</td>
<td>76.50 ± 3.91</td>
<td>106.57 ± 1.84</td>
<td>96.50 ± 1.24</td>
<td>89.96 ± 1.34</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>23.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>86.58 ± 2.56</td>
<td>76.50 ± 3.91</td>
<td>106.57 ± 1.84</td>
<td>96.50 ± 1.24</td>
<td>89.96 ± 1.34</td>
</tr>
<tr>
<td>LDH (U/L)</td>
<td>46.87 ± 2.16</td>
<td>37.73 ± 1.11</td>
<td>64.23 ± 1.62</td>
<td>56.03 ± 1.47</td>
<td>55.70 ± 1.51</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>86.87 ± 2.16</td>
<td>37.73 ± 1.11</td>
<td>64.23 ± 1.62</td>
<td>56.03 ± 1.47</td>
<td>55.70 ± 1.51</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>37.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

All data are mean of seven rats ± SE
* Significant (P < 0.05) as compared to the control group (N.C.).
# Significant (P < 0.05) as compared to the diabetic group (D.C.).

Changes in the Level of Lipid Peroxidation and Antioxidant Enzymes in the Liver of C. endivia treated Diabetic Rats:
Oxidative stress markers, liver CAT, SOD, GPx and GST activities as well as GSH and MDA levels of the different experimental groups are shown in Table 2. Administration of aqueous suspension of C. endivia leaves powder to normal rats had no effect on the studied oxidative stress markers in the liver, except SOD activity which decreased significantly (P<0.05), as compared to the control group. Intraperitoneal injection of STZ caused significant increase (P<0.05) in liver CAT, SOD activities and MDA level of rats, as compared to the normal control group (Fig. 2). On the other hand, STZ administration showed significant decrease (P<0.05) in the activities of liver GPx, GST and the level of GSH, as compared to the control group (Table 2 & Fig. 3). It is interesting to notice that the diabetic rats treated with aqueous suspension of C. endivia leaves powder showed significant decrease (P<0.05), of -9.51%, in liver SOD activity, and significant increase (P<0.05), of 38.46% and 23.06%, in GST activity and GSH level, respectively, as compared to the diabetic
group. Concerning the effect of glibenclamide drug on the diabetic rats, it seems that this drug ameliorates the alteration recorded in the liver antioxidant enzymes since, a significant decrease (P<0.05), of -29.17%, -12.33% and -11.96%, in the activities of liver CAT, SOD and the level of MDA, respectively, was as compared to the diabetic group. On the other hand, the activities of liver GPx, GST and the level of GSH of diabetic rats treated with glibenclamide drug showed significant increase (P<0.05), of 12.14%, 53.85% and 28.58%, respectively, as compared to the diabetic group (Table 2 & Fig. 3).

![Fig. 1: Serum AST, ALT, ALP and LDH activities (Mean ± SE) of experimental groups.](image)

a Significant (P<0.05) as compared with the normal control (N.C.).

b Significant (P<0.05) as compared with the diabetic control (D.C.).
Fig. 2: Liver CAT, SOD activity and MDA concentration (Mean ± SE) of experimental groups.

a Significant (P<0.05) as compared with the normal control (N.C.).

b Significant (P<0.05) as compared with the diabetic control (D.C.).

Fig. 3: Liver GPx, GST activity and GSH level (Mean ± SE) of experimental groups.

a Significant (P<0.05) as compared with the normal control (N.C.).

b Significant (P<0.05) as compared with the diabetic control (D.C.).
Table 2: Effect of treatment with aqueous suspension of *C. endivia* leaves powder or glibenclamide drug for 6 weeks on liver CAT, SOD, GPx and GST activities as well as GSH and MDA levels of diabetic rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Experimental groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N.C.</td>
</tr>
<tr>
<td>CAT (U/g tissue)</td>
<td>0.49 ± 0.10</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>46.94</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>8.33</td>
</tr>
<tr>
<td>SOD (U/g tissue)</td>
<td>655.89 ± 9.96</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>–</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>15.92</td>
</tr>
<tr>
<td>GPx (mU/ml)</td>
<td>84.26 ± 2.70</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>-15.82</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>–</td>
</tr>
<tr>
<td>GST (U/g tissue)</td>
<td>0.30 ± 0.02</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>-38.46</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>–</td>
</tr>
<tr>
<td>GSH (mg/g tissue)</td>
<td>46.59 ± 3.47</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>-26.50</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>–</td>
</tr>
<tr>
<td>MDA (nmol/g tissue)</td>
<td>168.77 ± 11.54</td>
</tr>
<tr>
<td>% of change from N.C.</td>
<td>-25.65</td>
</tr>
<tr>
<td>% of change from D.C.</td>
<td>–</td>
</tr>
</tbody>
</table>

All data are mean of seven rats ± SE.

*Significant (P < 0.05) as compared to the control group (N.C.).

**Significant (P < 0.05) as compared to the diabetic group (D.C.).

Discussion:

Streptozotocin-induced hyperglycemia has been described as a useful experimental model to study the activity of hypoglycemic agents (Junod et al., 1969). It damages the β-cells of the islets of Langerhans also hepatocytes, nephrons and cardiomyocytes (Selvan et al., 2008). STZ induces oxidative stress, which results from enhanced free radical formation and/or defects in antioxidants defense causes severe tissue damage and may lead to number of diseases like coronary artery disease, atherosclerosis, cancer and diabetes (Chakraborty and Das, 2010). The present study was designed to investigate the effect of aqueous suspension of *C. endivia* leaves powder or glibenclamide drug on some biochemical parameters in STZ-induced diabetic rats.

Aminotransferases (AST and ALT) mediate the catalysis of aminotransfer reactions and they are markers for clinical diagnosis of liver injury (Li et al., 2007). Alkaline phosphatase (ALP) is a hydrolase enzyme responsible for removing phosphate group from nucleotides and proteins, it is produced primarily in liver and brain (Han et al., 2006), and it is a marker of hepatic functions (Yoo et al., 2008). Lactate dehydrogenase (LDH) is a general indicator of acute or chronic hepatic damage, as well as determining organ, cell and tissue condition (Yoo et al., 2008). The present study demonstrated significant increase in the activities of serum AST, ALT, ALP, and LDH of diabetic rats as compared to the control group. This increase was mainly due to the leakage of these enzymes from the liver cytosol into the bloodstream (Navarro et al., 1993), which gives an indication on the hepatotoxic effect of STZ. Ohaeri (2001) also found that liver was necrotized in STZ-induced diabetic rats. Experimental diabetes induced by STZ in rats causes liver tissue damage, which is reflected in the increase of serum AST and various hepatic enzymes, such as ALT (Maiti et al., 2004). Khan et al. (2010) suggested that the elevation in serum AST and ALT activities in diabetic rats, could be related to excessive accumulation of amino acids (glutamate and alanine) in the serum of diabetic animals, as a result of amino acids mobilization from protein stores. Treatment with either aqueous suspension of *C. endivia* leaves powder or glibenclamide drug produced significant decrease in serum AST, ALT, ALP, and LDH activities, as compared to the diabetic group. It seems that *C. endivia* leaves powder or glibenclamide drug ameliorate the changes observed in the various hepatic enzymes of STZ-induced diabetic rats. The *C. endivia* leaves powder is better than glibenclamide drug in lowering serum ALT activity. Meanwhile, the effect of glibenclamide drug is more pronounced than that of *C. endivia* leaves powder in reducing the activity of serum ALP of diabetic rats. Aqueous suspension of *C. endivia* leaves powder and glibenclamide drug nearly appeared to have the same effect in decreasing the activity of serum AST and LDH of diabetic rats.

Superoxide dismutase (SOD) is one of the important antioxidant enzymes and scavenges the superoxide radicals by converting them to H₂O₂ and molecular oxygen (Selvan et al., 2008). Catalase (CAT) is a hemoprotein which catalyzes the reduction of hydrogen peroxide and protects the tissue from highly reactive hydroxyl radicals (Searle and Wilson, 1980). In the present study, hepatic SOD and CAT of the STZ-induced diabetic rats were increased significantly as compared to the control group. The elevation of liver SOD activity
may be due to the increase dismutation of superoxide anions, and the increase in the SOD activity may in turn protect CAT against enzyme inactivation by $\text{O}_2^-$ anions, as these anions have been shown to inactive CAT (Selvam and Anuradha, 1990). It is possible that the increase in oxygen radical during diabetes could increase CAT activity, which in turn would protect SOD inactivation by $\text{H}_2\text{O}_2$ and hence caused an increase in SOD activity. $\text{H}_2\text{O}_2$ is known to inactivate SOD (Bray et al., 1974). In accordance with our study, Cho et al. (2002) recorded an increase in CAT and SOD activities in the liver of STZ-induced diabetic rats. Regarding the effect of glibenclamide drug administration to diabetic rats there was significant reduction in hepatic SOD and CAT activities as compared to the diabetic group. However, treatment of the diabetic rats with aqueous suspension of C. endivia leaves powder produced only a significant decrease in their liver SOD activity.

Glutathione peroxidase (GPx), a selenium containing enzyme present in significant concentrations, detoxifies $\text{H}_2\text{O}_2$ to $\text{H}_2\text{O}$ through the oxidation of reduced glutathione (Bruce et al., 1982). It is sensitive to lower concentration of $\text{H}_2\text{O}_2$ (Bagri et al., 2009). Glutathione-S-transferase (GST) catalyzes the reduction of hydrogen peroxide and hydroperoxides to non-toxic products (Bruce et al., 1982). It catalyzes the conjugation of glutathione to a wide range of electrophiles and represents a protective mechanism against oxidative stress (Nowier et al., 2009). The present investigation revealed significant decrease in liver GPx and GST activities in the STZ-induced diabetic rats, as compared to the control group. In accordance with our results, Schettler et al. (1994) demonstrated that the reduced antioxidant production may be due to the increase in oxygen metabolites that causes a decrease in the activity of the antioxidant defense system. Moreover, Kennedy and Baynes (1984) reported that the decrease in antioxidant enzyme activity in diabetes mellitus may be due to non-enzymatic glycosylation of the enzymes. Again, Al-Wabel et al. (2008) suggested that the depletion of GSH content also may lower GST enzyme, because GSH is required as a substrate for GST activity. Treatment the diabetic rats with glibenclamide drug increased significantly the decline observed in the activities of liver GPx and GST, as compared to the diabetic group. Meanwhile, a significant increase in liver GST activity of diabetic rats was recorded after treatment with C. endivia leaves powder aqueous suspension, as compared to the diabetic group.

Reduced glutathione (GSH) normally plays the role of an intracellular radical scavengers and it is the substrate of many xenobiotic elimination reactions (Gregus et al., 1996). It protects the cellular system against the toxic effect of lipid peroxidation (Bagri et al., 2009). Viewed in conjunction with the report of Shabeer et al. (2009), the present reduction in hepatic GSH level following STZ administration may be due to its increased utilization by the hepatic cells, which could be result of decreased synthesis or increased degradation of GSH by oxidative stress in diabetes. Again, Veerapur et al. (2010) reported that the generation of oxygen radicals by the increase in the level of glucose caused tissue GSH depletion, which attempted to overcome the deleterious effects of lipid peroxidation. The present study revealed that liver GSH level of diabetic rats increased significantly after treatment with either C. endivia leaves powder aqueous suspension or glibenclamide drug, as compared to the diabetic group.

In diabetes, hypoinsulinaemia increased the activity of the enzyme fatty acyl coenzyme and coenzyme $A$ oxidase, which initiated $\beta$-oxidation of fatty acids resulting in lipid peroxidation (Baynes, 1995). The increase in oxygen free radicals in diabetes could be primarily due to the increase in the blood glucose level, which upon autoxidation generated free radicals, and secondarily due to the effect of the diabetogenic agent (streptozotocin or alloxan) (Szkudelski, 2001). Malondialdehyde (MDA) is one of the lipid peroxidation products frequently used to determine the oxidant/antioxidant balance in diabetic patient (Cheeseman and Slate, 1993). The present study revealed that the hepatic MDA level increased significantly after intraperitoneal injection of STZ as compared to the control group. In agreement with our study Punitha et al. (2006), Selvan et al. (2008) and Chakraborty and Das (2010) reported that the administration of STZ in rats elevated hepatic MDA level. The elevation in hepatic MDA may be due to the high concentration of lipid, which is one of the characteristic remarks in liver of diabetic rats, which resulted in the activation of NADPH dependent microsomal lipid peroxidation in liver (Patel et al., 2009). Treatment with glibenclamide drug showed significant decrease in liver MDA level of diabetic rats. But after treatment with C. endivia leaves powder aqueous suspension, the level of liver MDA of diabetic rats showed insignificant decrease, as compared to the diabetic group. The results suggest that aqueous suspension of C.endivia leaves powder or glibenclamide drug may effectively normalize the impaired antioxidants status in STZ-induced diabetes. Moreover, glibenclamide drug may be more powerful free radical scavenger than C. endivia leaves powder.

In conclusion, the results obtained clearly indicate the role of oxidative stress in induction of diabetes and suggest the ameliorative effect of C.endivia leaves powder aqueous suspension, similar to the diabetic drug (glibenclamide) at least in such animal model. However, further studies are necessary to isolate and identify the principle components of C. endivia leaves powder and elucidate the mechanisms of their action.
REFERENCES


