Short Communication

Effects of cinnamon (Cassia zelynicum) on diabetic rats

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The purpose of this study is to show the effects of crude cinnamon extract on the diabetic rats. Diabetes mellitus manifests itself in a wide variety of complications and the symptoms of the disease are multifactorial. The sugar level and lipid profile were investigated in plasma of normal and alloxan-induced diabetic rats treated with cinnamon for four weeks. Diabetic rats exhibited an increase in the levels of sugar, cholesterol, triglycerides and low density lipoprotein (LDL), and a decrease in the level of high density lipoprotein (HDL). The administration of cinnamon showed a decrease of 29% in sugar level, 24% in cholesterol, 19% in triglycerides, 26% in LDL, and increase of 20% in (HDL). In conclusion, the findings of this study indicate that the administration of cinnamon showed better lipid profile as well as decreases in the sugar level in both normal and diabetic rats.

Key words: Cinnamon, diabetes, lipid profile.

INTRODUCTION

A number of spices and herbs have a long history of traditional use in treating elevated blood sugar levels (Anderson, 2004). One of such compound that has recently been the subject of intense research is cinnamon, a compound that is granted as generally recognized as safe (GRAS) status by the United States Food and Drug Administration (FDA). Cinnamon has been shown to be generally safe when ingested and have many pharmacological properties, such as antioxidants activity and antibacterial effects (Imparl et al., 1998; Shan, 2005).

Recently, Richard Anderson and his team at the US Department of Agriculture’s Human Nutrition Research Center in Beltsville, Maryland, discovered the scientific evidence that demonstrates how cinnamon serves as an important antioxidant, and is beneficial in the prevention and control of glucose intolerance and diabetes (Lopez et al., 2005). Oxidative stress generated by hyperglycemia and hyperlipidemia is regarded as an important mediator of diabetic complications. The presence of free radicals and the simultaneous decline of antioxidant defense capabilities observed in diabetic patients could promote the development of diabetic complications (Godin et al., 1988). Alloxan has been proposed to act as a diabetogenic agent due to its ability to destroy pancreatic β-islets cells, which was possibly by increasing free radical accumulation. Diabetes represents a state of increased lipid peroxidation and reduced antioxidant reserve (Panneerselvam and Govindasamy, 2004).

This study evaluates the antioxidant activity of cinnamon in normal and alloxan induced diabetic rats. Moreover, the role of cinnamon in sugar level and lipid profile change was investigated.

MATERIALS AND METHODS

Animals

Sixty male albino rats weighing between 160 and 180 gm were procured from Department of Medical Technology, Zarqa Private University, Jordan. The animals were housed in a well ventilated 12 h light and dark cycle. The animals were divided into 4 equal groups: Group I, normal rats as control; group II, normal rats orally administered with cinnamon (0.16 gm/kg); group III, alloxan treated animals (150 mg/kg intraperitoneally) to be diabetic rats; group IV, diabetic rats that are treated with alloxan and orally administered with cinnamon (0.16 gm/kg).
Table 1. Depicts the level of plasma glucose and lipid profile at zero time.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sugar</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL</th>
<th>LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>90.2 ± 5.2</td>
<td>95.1 ± 3.8</td>
<td>85.21 ± 3.41</td>
<td>67.3 ± 2.2</td>
<td>53.51 ± 4.2</td>
</tr>
<tr>
<td>2</td>
<td>83.5 ± 4.21</td>
<td>86.72 ± 3.1</td>
<td>75.62 ± 5.7</td>
<td>72.45 ± 5.9</td>
<td>58.28 ± 3.2</td>
</tr>
<tr>
<td>3</td>
<td>301.2 ± 5.6</td>
<td>179.6 ± 3.1</td>
<td>190.3 ± 11.2</td>
<td>45.71 ± 2.1</td>
<td>83.41 ± 7.3</td>
</tr>
<tr>
<td>4</td>
<td>298.52 ± 4.2</td>
<td>172.62 ± 5.4</td>
<td>215.91 ± 7.3</td>
<td>49.82 ± 2.8</td>
<td>78.61 ± 5.17</td>
</tr>
</tbody>
</table>

Table 2. Depicts the level of plasma glucose and lipid profile at the end of 4 weeks.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sugar</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL</th>
<th>LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>92.5 ± 2.2</td>
<td>90.9 ± 1.5</td>
<td>80.26 ± 1.47</td>
<td>60.3 ± 4.7</td>
<td>55.21 ± 3.3</td>
</tr>
<tr>
<td>2</td>
<td>80.41 ± 3.1</td>
<td>81.52 ± 3.1</td>
<td>70.12 ± 5.8</td>
<td>72.15 ± 8.2</td>
<td>50.31 ± 2.2</td>
</tr>
<tr>
<td>3</td>
<td>293.81 ± 9.6</td>
<td>199.4 ± 2.9</td>
<td>215.72 ± 14.1</td>
<td>56.21 ± 2.5</td>
<td>83.61 ± 3.1</td>
</tr>
<tr>
<td>4</td>
<td>210.42 ± 9.2**</td>
<td>152.82 ± 6.1*</td>
<td>175.71 ± 6.3*</td>
<td>70.52 ± 2.5*</td>
<td>61.61 ± 3.1*</td>
</tr>
</tbody>
</table>

** Highly significant at (p < 0.01). * Significant at (p < 0.05).

Cinnamon extraction

50 gm of grind cinnamon were soaked in 150 ml hot water (88°C) in water bath for 6 h. Then filtered by capron silica cloth 150 µ and the filtrate were stored in dark bottles in the refrigerator at (4°C). These procedures were repeated each week.

Blood sampling

Blood was collected after three days from alloxan treatment and zero time was considered. Moreover, blood was taken at zero time and 4 weeks from eyes of all groups in heparinized tubes. Plasma was separated and kept in freezer till the time of assay.

Biochemical analysis

The following analyses were carried out: Glucose, Cholesterol, Triglycerides, Low and high density lipoprotein using kits from Syrbio, France.

Statistical analysis

Collected data were tabulated and needed statistical analyses were done using the computer data processing (SPSS, version 12). A probability value P of < 0.05 was considered to be statistically significant.

RESULTS

Table 1, shows the level of plasma glucose, cholesterol, triglyceride, HDL and LDL in both control and experimental rats at zero time. Table 1 also showed that the diabetic rats marked an elevation in glucose concentration and lipid profile.

Cinnamon has no significant influence on plasma glucose level and lipid profile of normal rats.

Table 2 shows the mean value of the sugar and lipid profile after using cinnamon for 4 weeks. Their reduction of 29% in sugar level, 24% in cholesterol, 19% in triglyceride, 26% in LDL and increase of 20% in HDL were significant at p < 0.01 and p < 0.05, respectively.

Since the cinnamon is one of the dietary components that are being used daily in our food products, and it is well known that it is safe for human consumption, we recommended that it can be applied on human volunteer diabetic patients in forwarding this work to the next step.

DISCUSSION

The world is facing an explosive increase in the incidence of diabetes mellitus and cost effective complementary therapies are needed. Although insulin has become one of the most important therapeutic agents known to medicine, there is a continuing effort to find insulin substitutes, secretagogues, or sensitizers from synthetic or plant sources for the treatment of diabetes (Pushparaj et al., 2001; Bhandari et al., 2005).

In the present study, the aqueous extract of cinnamon was investigated for its antidiabetic activity in diabetic rats. Animal models of diabetes are increasingly being used for pathophysiology and pharmacological studies of diabetes mellitus. Advantages of animal studies in the examination of alternative medicines and their efficacy include the ability to define experimental conditions more tightly and to undertake more detailed studies of the biologic effects of the agents being used (Batey et al., 2005).

In our study, the intraperitoneal administration of alloxan to normal rats effectively induced diabetes as reflected by the increase in glycosuria, hyperglycaemia and hypoinsulinaemia. Cinnamon treatment showed significant hypoglycaemic and antihyperglycaemic effects. The experimental results indicated that cinnamon exhibited the lowering of blood glucose and lipid profile.
both in normal and diabetic rats.

Our findings also agree with the recent studies of Khan et al. (1990) who isolated a factor from cinnamon, which could affect a three-fold increase in glucose metabolism in rat epididymal fat cells and attributed it to the presence of MHCP. Cinnamon extract can stimulate autophosphorylation of the insulin receptor and can inhibit protein tyrosine phosphatase-1 (PTP-1), which inactivates insulin receptor in the adipocytes. It was suggested that cinnamon could affect protein phosphorylation-dephosphorylation reactions in the intact adipocytes (Imparl et al., 1998). Cinnamon was the most bioactive product among the 49 botanic products tested for the in-vitro effects on insulin-dependent metabolism of glucose in the adipocytes (Broadhurst et al., 2000).

Oral treatment of normal rats with cinnamon extract enhances the glucose utilization in-vivo in a dose-dependent manner and potentiates the insulin-stimulated tyrosine phosphorylation of insulin receptor substrate (Qin et al., 2003).

Qin et al. (2004) also found that cinnamon extract prevents the development of insulin resistance in HFD rats. They attributed this to the activation of the insulin signaling possibly via the nitric oxide pathway in the skeletal muscle.

Improved insulin action in CBEt-treated rats could be responsible for the regulation of lipid metabolism, and hence we observed normal plasma lipid profile in them. However, studies show that cinnamon might also have a direct role in lipid metabolism. For example, cinnamon bark powder at different doses (1, 3 and 6 g/day) prevents hypercholesterolaemia and hypertriglyceridaemia and lowers the levels of free fatty acids and triglycerides in plasma of type 2 diabetic subjects as a result of its strong lipolytic activity (Khan et al., 2003). Cinnamate, a phenolic compound found in cinnamon bark and other plant materials, lowers cholesterol levels in high fat-fed rats by inhibiting hepatic 5-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase activity (Lee et al., 2003).

Our findings provide evidence for the therapeutics potential of cinnamon in the treatment of insulin resistant states.

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REFERENCES


