COMPARATIVE STUDY TO GINSENG AND CINNAMON WATER EXTRACT ON DIABETIC ADULT MALE ALBINO RAT

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ABSTRACT

Background: Constituents of ginseng root produce immunomodulatory, vasodilatory, anti-inflammatory, antioxidant, anti-aging, anticancer, anti-fatigue, anti-stress and anti-depressive effects. Ginseng treatment improves mitochondrial and hypothalamus-pituitary-adrenal axis function and increases anabolic hormone secretion. Water-soluble cinnamon compounds stimulate the autophosphorylation of the insulin receptor and inhibit phosphotyrosine phosphatase, an enzyme functioning in the dephosphorylation of the insulin receptor.

Objective: Comparing the effects of ginseng and aqueous extract on diabetic adult male albino rat.

Materials and Methods: Eighty rats of local strain were used for studying these effects. The animals were divided equally into ten equal groups: three of them were non-diabetic received either distilled water, ginseng or cinnamon. The 4th group was diabetic received distilled water. The remaining six groups were diabetic either treated or pretreated with ginseng or cinnamon or both. The experimental procedure continued for one month. At the end of the experiment, body weight and rat tail systolic blood pressure were measured, then blood samples were taken for blood glucose, HbA1c, total cholesterol, triglycerides, high density lipoprotein, low density lipoprotein, and CRP levels. Specimens from liver were taken for histopathological studies.

Results: Results of the present study revealed that diabetic group receiving distilled water showed significant elevation of blood glucose, HbA1c, total cholesterol, triglycerides, high density lipoprotein, low density lipoprotein, and CRP levels. Hepatocytes were markedly infiltrated with fat, and showed marked reduction in mitochondrial and glycogen content. Treatment with ginseng caused significant improvement in HDL and CRP levels. Treatment with cinnamon caused significant improvement in cholesterol TAGs, CRP, systolic blood pressure with remarkable improvement in mitochondrial and glycogen contents of hepatocytes which were also less degenerated than the diabetic control group. Pretreatment with either ginseng, cinnamon or both showed a protective effect against alloxan-nicotinamide induced diabetes. Conclusion: Both ginseng and cinnamon could be of great value in diabetic management and able to alter the different mechanisms included in the pathogenesis of diabetic complications namely hyperlipidemia, oxidative stress and stimulating inflammatory processes. Also, both agents showed hepatic protective effects against diabetic induced hepatic injury.

Key words: Diabetes, Ginseng, Cinnamon, lipid profile.

INTRODUCTION

Diabetes-induced tissue damage affects a particular subset of cell types, i.e. blood vessel endothelial cells, mesangial cells in the renal glomerulus, neurons and Schwann cells in peripheral nerves. Most cells are able to reduce the transport of glucose inside them when they are exposed to hyperglycemia so that their internal glucose concentration stays constant. In contrast, the cells damaged by hyperglycemia are those that cannot do this efficiently (Kaiser et al., 1993).
The bioactive ingredients in ginseng root include more than 60 ginsenosides as well as polysaccharides, fatty acids, oligopeptides and polyacetylenic alcohols (Qian et al., 2006). Ginseng treatment improves mitochondrial and hypothalamus-pituitary-adrenal axis function and increases anabolic hormone secretion (Jung et al., 2016). Anti-oxidative and anti-inflammatory effects of ginseng were reported. Ginseng extract induces elevation of the important free radicals scavengers; catalase and superoxide dismutase enzymes (Saw et al., 2010).

Cinnamon improves the serum glucose and lipid levels in type 2 diabetic subjects (Shen et al., 2010). The compounds found in cinnamon have insulin-potentiating properties and may be involved in the alleviation of the signs and symptoms of diabetes, and coronary vascular diseases related to insulin resistance (Khan et al., 1990).

The present work was directed to study the effect of ginseng and aqueous cinnamon extract on blood glucose, HbA1c, lipid profile, CRP, body weight and systolic blood pressure, as well as their effects on general morphology, mitochondrial and glycogen content in liver specimens in alloxan-nicotinamide-induced diabetes mellitus.

**MATERIALS AND METHODS**

**I- Materials**

**Animals:** Eighty adult male albino rats of a local strain were used as an animal model for this study. Their ages were 8 weeks and their weight 120 – 150 g. Animals were purchased from ACMA Pharmaceuticals Company. They were kept in suitable cages (20x32x20 cm for every 4 rats) at room temperature with the natural light-dark cycle. They were maintained on a standard diet of commercial rat chow and tap water. They were kept for 10 days for the adaptation to the new environment before starting the experiment in physiology laboratory, Al-Azhar Faculty of Medicine.

**Drugs:**

1. **Alloxan** (Nile Pharmaceuticals Company-Egypt): It was dissolved in 0.9% NaCl solution and given IP in a dose of 90 mg/kg BW (Szkudelski, 2001).

2. **Nicotinamide** (Sigma Aldrich Pharmaceuticals Company-USA): It was dissolved in 0.9% NaCl solution and given IP in a dose of 110 mg/kg BW (Madkor et al., 2011).

3. **Ginseng** (Jamieson-Canada): Each tablet (500 mg) was crushed into powder. This powder was then dissolved in 20 ml of distilled water and mixed well using magnetic stirrer to help maximum dissolution. The solution was then filtered through chess cloth. The weight of this cloth was determined before by using a sensitive balance. The chess cloth was then left to dry and weighed again to calculate the un-dissoluted fraction of the ginseng powder (about 100 mg for each tablet).

   Depending on the basis of the previous weights, the concentration of ginseng in the solution was 20 mg/ml. Ginseng then was given orally by gavaging in a dose of 100 mg/kg BW daily (Gupta et al., 2001).

4. **Cinnamon extract:** Cinnamon bark was purchased from the local market.
The bark was left to dry and finely powdered in an electrical blender. Ten grams of finely-powdered cinnamon was mixed with 100 ml of distilled water and kept in a water bath at 60°C for two hours, then filtered by cheesecloth. The extract was diluted with distilled water (one part cinnamon extract and 10 parts water) and given to rats by gavaging in a dose of 2 ml/rat daily (Kannappan et al., 2006).


II- Methods

The animals were divided into ten equal groups as follows:

I- Normal control group received 0.5 ml distilled water by gavaging daily.

II- Non-diabetic ginseng-treated group received ginseng at a dose of 100 mg/kg body weight (BW) by gavaging daily (Gupta et al., 2001).

III- Non-diabetic cinnamon-treated group received aqueous cinnamon extract at a dose of 2 ml/rat by gavaging daily (Kannappan et al., 2006).

IV- Diabetic group received 0.5 ml distilled water by gavaging daily.

V- Diabetic ginseng-treated group received ginseng at a dose of 100 mg/kg BW by gavaging daily.

VI- Diabetic group pretreated with ginseng received ginseng at a dose of 100 mg/kg body weight (BW) by gavaging daily before induction of diabetes.

VII- Diabetic cinnamon-treated group received cinnamon extract at a dose of 2 ml/rat by gavaging daily.

VIII- Diabetic group pretreated with cinnamon received cinnamon at a dose of 2 ml/rat by gavaging daily before induction of diabetes.

IX- Diabetic ginseng and cinnamon-treated group received ginseng at a dose of 100 mg/kg BW and cinnamon extract at a dose of 2 ml/rat by gavaging daily.

X- Diabetic group pretreated with ginseng and cinnamon received ginseng at a dose of 100 mg/kg BW and cinnamon at a dose of 2 ml/rat by gavaging daily before induction of diabetes.

The experimental procedure continued for one month.

Sequence of events:

Rats were starved for 24 hours in specific cages with a perforated floor in order to avoid coprophagia. In the next day, nicotinamide was dissolved in 0.9% NaCl. Each rat was weighed and injected with the nicotinamide intraperitoneally at a dose of 110 mg/kg BW (Madkor et al., 2011). After about 20 minutes, alloxan was dissolved in 0.9% NaCl and injected intraperitoneally at a dose of 90 mg/kg BW (Szkudelski, 2001). Just before alloxan injection, 2ml of glucose (5%) were given orally. After 48 hours, blood samples were taken from tail vein for blood sugar estimation. Rats with blood sugar higher than 200 mg/dl were considered diabetic.

At the end of the experiment, body weight was recorded. Also, systolic blood pressure was recorded using rat tail systolic blood pressure apparatus (Harvard-USA). Blood samples were collected from the retro-orbital venous
plexus by using a heparinized capillary tube (about 0.75 – 1.0 mm internal diameter) inserted in the medial canthus. The collected blood samples were kept in clean graduated plastic centrifuge tubes containing EDTA. About half milliliter of the blood was taken in another plastic tube and stored at 4°C till being used for estimating blood HbA1c. The remaining blood was centrifuged at 5000 rotations per minute for about 15 minutes to separate the serum. Serum was sucked out into Eppendorf tubes, and stored frozen at -20°C till used for the measurement of:

- Blood glucose level (Braham and Trinder, 1972)
- Glycated Hemoglobin - HbA1c (Zander et al., 1984)
- Total cholesterol (Allain et al., 1974)
- Triglycerides –TAGs (Fossati and Prencipe, 1982)
- High Density Lipoprotein cholesterol – HDLc (Groove, 1979)
- Low Density Lipoprotein cholesterol – LDLc (Friedewald et al., 1972)
- C Reactive Protein - CRP (Urdal et al., 1992)

**Statistical analysis:** The computer program SPSS version "17" was used to perform the statistical analysis for:

- Descriptive statistics in studied groups (means ± standard deviations).
- Differences between different groups using one way ANOVA (Analysis Of Variance) test.
- Multiple comparisons between each group and another by using the "Post Hoc least significant difference [LSD]" multiple comparison test.

The difference was considered significant when P value was less than 0.05.

## RESULTS

Blood glucose level was significantly lower in the diabetic pretreated group whether with ginseng or cinnamon or both together when compared with the diabetic non-treated group. Changes in blood glucose levels were more or less parallel to changes in Hb A1c levels in different groups. Higher levels of blood glucose were associated with higher levels of HbA1c and vice versa (Figure 1).

There was significant improvement in cholesterol and TAGs levels in the diabetic group treated with cinnamon when compared with the diabetic non-treated group. HDLc showed significant elevation in diabetic ginseng treated group when compared with the diabetic-non treated group. Changes in lipid profile parameters were more or less concomitant in studied groups, i.e. elevated cholesterol was associated with elevated LDL and TAGs levels but decrease in HDL level and vice versa (Figure 2).
CRP showed significant improvement in cinnamon-treated diabetic group when compared with the diabetic non-treated group. Also, CRP levels were significantly lower in the ginseng, cinnamon, and ginseng-cinnamon-pretreated groups when compared with the diabetic non-treated group. Body weight was significantly higher in the diabetic groups pretreated with either ginseng or cinnamon or both when compared with the diabetic non-treated group. Systolic B.P showed significant improvement in the treated and pretreated groups either with ginseng or cinnamon or both when compared with the diabetic non-treated group (Table 1).

**Table (1):** Changes in body weight, systolic blood pressure and CRP (Mean ± S.D).

<table>
<thead>
<tr>
<th>Groups</th>
<th>CRP (mg/L)</th>
<th>Body weight (grams)</th>
<th>Systolic blood pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group &quot;I&quot; Normal (received distilled water by gavaging)</td>
<td>2.53 ± 0.3</td>
<td>166.5 ± 23.4</td>
<td>110.4 ± 7.4</td>
</tr>
<tr>
<td>Group &quot;II&quot; Normal (received ginseng by gavaging)</td>
<td>2.49 ± 0.26</td>
<td>171.0 ± 21.4</td>
<td>113.6 ± 10.3</td>
</tr>
<tr>
<td>Group &quot;III&quot; Normal (received cinnamon by gavaging)</td>
<td>2.39 ± 0.12</td>
<td>174 ± 21.8</td>
<td>104.0 ± 7.5</td>
</tr>
<tr>
<td>Group &quot;IV&quot; Diabetic (received distilled water by gavaging)</td>
<td>3.13 ± 0.45</td>
<td>136.0 ± 18.5</td>
<td>52.0 ± 4.5</td>
</tr>
<tr>
<td>Group &quot;V&quot; Diabetic (received ginseng by gavaging)</td>
<td>3.13 ± 0.31</td>
<td>139.0 ± 15.6</td>
<td>93.0 ± 2.5 *</td>
</tr>
<tr>
<td>Group &quot;VI&quot; Diabetic (pretreated with ginseng)</td>
<td>2.63 ± 0.13 *</td>
<td>166.4 ± 21.2 *</td>
<td>127.5 ± 14.5 *</td>
</tr>
<tr>
<td>Group &quot;VII&quot; Diabetic (received cinnamon by gavaging)</td>
<td>2.5 ± 0.27 *</td>
<td>140.0 ± 7.9</td>
<td>87.1 ± 11.8 *</td>
</tr>
<tr>
<td>Group &quot;VIII&quot; Diabetic (pretreated with cinnamon)</td>
<td>2.45 ± 0.26 *</td>
<td>163.0 ± 22.1 *</td>
<td>103.8 ± 14.6 *</td>
</tr>
<tr>
<td>Group &quot;IX&quot; Diabetic (received ginseng and cinnamon by gavaging)</td>
<td>3.35 ± 0.32</td>
<td>154.8 ± 32.9</td>
<td>120.6 ± 17.7 *</td>
</tr>
<tr>
<td>Group &quot;X&quot; Diabetic (pretreated with ginseng and cinnamon)</td>
<td>2.42 ± 0.24 *</td>
<td>149.2 ± 22.7 *</td>
<td>138.0 ± 16.3 *</td>
</tr>
</tbody>
</table>

(*): significant difference when compared with diabetic non-treated group IV

**DISCUSSION**
The metabolic abnormalities of diabetes cause mitochondrial superoxide overproduction. This increased superoxide production is the central and major mediator of diabetes-induced tissue damage, causing the activation of five pathways involved in the pathogenesis of diabetic complications. These pathways increase flux of glucose and other sugars through the polyol pathway (Lee and Chung, 1999), increased intracellular formation of advanced glycation end products (AGEs) (Candido et al., 2003), increased expression of the receptor for AGEs and its activating ligands, activation of protein kinase (PK) C isoforms (Brownlee, 2005), and finally overactivity of the hexosamine pathway (Du et al., 2000). Also, increased oxidative stress leads to direct inactivation of two antiatherosclerotic enzymes, i.e. nitrous oxide synthase (NOS) and prostacyclin synthase (Giacco and Brownlee, 2010).

In the present study, there was a significant increase of glucose and HbA1c levels in diabetic group (IV) when compared with the control group (I). These results were in agreement with the findings of Szkudelski (2001), Elsner et al. (2002) and Eleazer (2003) who found that male albino rats given a single injection of alloxan showed an elevated random blood glucose level, decreased serum insulin level, and developed diabetes mellitus. This was due to production of excess reactive oxygen species (ROS) in β-cells of the pancreas in alloxan-treated animals. These ROS produced damage of these cells. This was compatible with Bromme et al. (2001) who stated that β-cell damage induced by alloxan occurs through the noxious oxygen free radicals such as O₂⁻, H₂O₂ and malondialdehyde (MDA). Also, Green et al. (2004) mentioned that reactive oxygen species produced by alloxan treatment lead to breakdown of DNA strands. Such damaged DNA activates nuclear poly synthetase which depletes the cellular pool of NAD⁺, resulting in β-cell damage.

In the present study, there was a significant increase of cholesterol, TAGs and LDL and significant reduction in HDL in diabetic group (IV) when compared with the control group (I). These results were compatible with the findings of Thomson et al. (2007) and Ali & Agha (2009) who found that cholesterol, TAGs and LDL levels showed significant elevations in diabetic animals when compared with normal ones. Also, Bennion & Grundy (1977), Laakso (1996) and Abbate & Brunzell (1990) reported that elevated serum cholesterol and TAGs levels occur in both type I and type II diabetes, and tend to fall toward normal with control of hyperglycemia.

CRP is one of the main inflammatory markers that are used and have been proved to provide prognostic information on the outcome and progression of the disease in diabetic patients (Tousoulis et al., 2013). Our study showed that C-reactive protein "CRP" levels increased significantly in diabetic group IV when compared with the normal group I. This result was in agreement with the results of Schram et al. (2005), Goyal et al. (2008), Sedigheh et al. (2011), Esser et al. (2014) and Rahman et al. (2016) who reported that CRP level was significantly higher in diabetic rats than in normal control rats. Yoshida et al. (2006) stated that CRP, an acute-phase reactant mainly produced by the liver in response to pro-inflammatory
cytokines, is elevated in diabetes and contributes to the development and progression of atherosclerosis. However, the molecular mechanism underlying the elevation of CRP in diabetes is not fully understood. Panveloski-Costa et al. (2016) reported higher CRP levels in diabetic rats and stated that a chronic low-grade inflammation is a common feature in diabetes. Increase in pro-inflammatory molecules as interleukins and TNF-α, and alterations in their regulation do exist in states of diabetes mellitus (DM) this in turn will lead to higher levels of CRP.

In addition, CRP elicits endothelial dysfunction, pro-inflammatory reactions and vascular smooth muscle cell proliferation, thereby directly contributing to the development and progression of atherosclerosis (Jialal et al., 2004). Moreover, increased CRP level is not only independent predictor for the vascular complications which may associate DM, but also it is considered a predictor for the development of type II DM in apparently healthy women, supporting the hypothesis that subclinical inflammation is an underlying factor in the pathogenesis of type II DM (Hu et al., 2004).

In our study, CRP was significantly lower in diabetic group treated with cinnamon (VII) when compared with the diabetic untreated group (V). This result was consistent with Azab et al. (2011), Askari et al. (2014) and Tangvarasittichai et al. (2015) whose study demonstrated the health benefits of cinnamon in diabetic patients such as reduction of glucose, malondialdehyde (MDA), CRP, improvement of insulin level, insulin resistance, β-cell function, and increasing insulin sensitivity.

The reduction of CRP by cinnamon is mediated by its effects on cytokines, in particularly IL-1, TNF-α, and IL-6, which are the main inducers of this acute phase response. The antioxidant activity of cinnamon is responsible for inhibition of induced production of such cytokines with subsequent anti-inflammatory activity (Hartel et al., 2004). Diabetes is an inflammatory disease. Inflammation contributes in the development of diabetes and then the inflammatory process continues contributing in the development of its complications (Xie and Du, 2011).

Our study also showed a significant decrease in BW in the diabetic group (IV) when compared with the control group (I). This result was compatible with Stanely et al. (2000) and Ene et al. (2007) who reported that body weight decreased in alloxan diabetic rats. Frier and Fisher (2006) stated that profound insulin deficiency causes unrestrained lipolysis and proteolysis result in weight loss. This is because insulin is not only a glucose lowering hormone but it is also a fat storage hormone.

If there is marked insulin deficiency, there is no fat storage. So, it is impossible to gain weight. Because cells are starving with no insulin to let glucose in, the body begins breaking down fat and muscle in an attempt to feed the cells. Obviously, this causes weight loss. Cooke and Plotnick (2008) reported that, with further insulin deficiency, there is an increase in lipolysis from fat cells as well as protein breakdown, an exaggeration of the normal fasting state designed to provide alternative sources of fuel. These mechanisms, along with the caloric loss from glucosuria, result in weight loss.
In the present study, treatment with ginseng causes significant elevation of HbA1c in group V (diabetic treated with ginseng) when compared with the diabetic untreated group (IV). This hyperglycemic effect was also found in group IX (diabetic treated with both ginseng and cinnamon) as when comparing this group with the diabetic untreated group (IV). HbA1c was significantly higher. On comparing this group (group IX) with group VII (diabetic treated with cinnamon alone), blood glucose and HbA1c levels were lower in group treated with cinnamon alone (VII) than the group treated with both ginseng and cinnamon (IX).

Ginseng has insulin secreting and sensitizing effect (Cho et al., 2006; Jeon et al., 2013 and Gun-Sub et al., 2015). This tends to lower blood glucose levels in ginseng-treated diabetic rats. However, Moon et al. (2015) reported that blood glucose levels improved in ginseng-treated diabetic rats when ginseng was given in doses of 200 and 300 mg/kg BW, with better improvement in higher dose. Treatment with 100 mg/kg of ginseng (similar to the dose in our study) has not improved blood sugar level.

Nocerino et al. (2000) stated that ginseng is considered a tonic or adaptogenic that enhances physical performance. The adaptogenic properties of ginseng are believed to be due to its effects on hypothalamic-pituitary-adrenal axis, resulting in elevated plasma corticotrophin and corticosteroids levels. So, ginseng has 2 important endocrinial effects, i.e. it increases the production of insulin from pancreatic β cells, and stimulates the release of corticotrophin hormone from anterior pituitary with subsequent increase in corticosteroid level. This state of hypercortisolism is associated with insulin resistance and worsens diabetes mellitus (Joshua et al., 2015). Predominance of one of these two endocrinial effects appears to be dose-dependent. Insulin secreting effect is marked in higher doses of ginseng (Moon et al., 2015).

In the present study, the diabetic cinnamon-treated group (VII) showed marked improvement in blood glucose and HbA1c levels when compared with the diabetic untreated group (IV). Also, the diabetic cinnamon-treated group showed significant improvement in both blood glucose and HbA1c levels when compared with the diabetic ginseng-treated group. These results were in agreement with the finding of Mang et al. (2006) who reported that cinnamon extract has moderate effect in reducing plasma glucose. Hafizur et al. (2015) reported the anti-diabetic activity of cinnamic acid, a pure compound from cinnamon, and stated that cinnamic acid decreased blood glucose levels in diabetic rats in a time- and dose-dependent manner. The improvement was comparable to that of standard drug glibenclamide. Cinnamic acid significantly enhanced glucose-stimulated insulin secretion from pancreatic islets. Similar results were also reported by Onderoglu et al. (1999) and Jarvill-Taylor & Graves (2001) who reported that cinnamon contains some constituents as cinnamon oil, euoginol, thyme oil and cumarin which can enhance insulin secretion, reinforce insulin performance and improve insulin receptor phosphorylation. Shen et al. (2014) demonstrated that
cinnamon extract ameliorates type I induced diabetes in rats through the up-regulation of glucose transporter 4 translocation in both muscle and adipose tissues. Shihabudeen et al. (2011) demonstrated one of the mechanisms by which cinnamon bark extract exerts a hypoglycemic effect by inhibiting α-glucosidase leading to suppression of postprandial hyperglycemia. Cinnamon extract could be used as a potential nutraceutical agent for treating postprandial hyperglycemia.

In our study, there was a significant improvement in both cholesterol and TAGs levels in diabetic cinnamon-treated group (VII) when compared with the diabetic-untreated group (IV). This lipid lowering effect of cinnamon was also reported by Khan et al. (2003), Kang et al. (2006) and Vafa et al. (2012). Cinnamon extract exerts a blood glucose-suppressing and lipid lowering effect by increasing insulin secretion, improving insulin sensitivity and slowing absorption of carbohydrates in the small intestine (Kang et al, 2006). Lee et al. (2004) and Ping et al. (2010) reported that supplementation with cinnamon resulted in significantly lower cholesterol and triglyceride levels. The lipid lowering effect of cinnamon was also proved by Cao et al. (2010) who reported that cinnamon water extract regulates the expression of multiple genes in adipocytes, and this regulation could contribute to the potential health benefits of cinnamon.

Our study showed a hypolipidemic effect of ginseng evidenced by:

1. Treatment with cinnamon alone caused detectable improvement in LDL level in group VII (diabetic treated with cinnamon) in comparison with the diabetic untreated group (IV), whereas treatment with both ginseng and cinnamon caused significant reduction of LDL level in group IX (diabetic treated with both ginseng and cinnamon) when compared with the diabetic-untreated group (IV).

2. Group V (diabetic treated with ginseng) showed significant improvement in HDL level when compared with the diabetic-non treated group (IV).

3. Group X (diabetic pretreated with both ginseng and cinnamon) showed significant reduction in LDL level when compared with group III (non-diabetic treated with cinnamon alone).

The lipid improving effect of ginseng was recorded by Cho et al. (2006), Liu et al. (2013) and Murthy et al. (2014) who reported that treatment with ginseng improves insulin sensitivity and hence its action on tissues with subsequent improvement in lipid profile.

In our study, the systolic blood pressure of the diabetic control group (IV) was significantly lower than the systolic blood pressure of the normal group (I). This result was consistent with the finding of Jackson and Carrier (1983) who reported that diabetic rats were hypotensive when compared with control rats. Also, Chang & Lund (1986) and Fazan et al. (1999) reported that baseline blood pressure of diabetic rats was significantly lower when compared to age-matched control rats. Borges et al. (2006) stated that hypotension has been described in diabetic rats and attributed to: 1) decreased cardiac output 2) hypovolemia due to osmotic diuresis 3) impairment...
of sympathetic innervation of heart and vessels. In our study, there was significant elevation of systolic blood pressure in group V (diabetic treated with ginseng), group VI (diabetic pretreated with ginseng) and group X (diabetic pretreated with both ginseng and cinnamon) when compared with the diabetic untreated group (IV), i.e. almost all groups treated with ginseng showed elevation in systolic blood pressure level.

This hypertensive effect of ginseng was recorded by Buettner et al. (2006) who stated that there is concern about use of ginseng in individuals with diabetes due to possible adverse effects, including raising blood pressure to hypertensive levels. Our results were also concomitant with Nocerino et al. (2000) and David and Traci (2003) who stated that hypertension is one of the documented effects of ginseng. This blood pressure elevating effect could be attributed to the effect of ginseng on hypothalamo-pituitary-adrenal axis. Ginseng stimulates the release of ACTH from anterior pituitary and glucocorticoids from suprarenal cortex (Nocerino et al., 2000).

Both adrenocorticotrophin (ACTH) and glucocorticoids raise blood pressure in man and animals (Connell et al., 1987). Increase in cardiac output is an aiding factor in cortisol-induced blood pressure rise but the precise role is the increased pressor responsiveness, particularly to catecholamines (Whitworth et al., 1995). Wei-Yi et al. (2015) stated that ginseng has a general stimulatory effect on CNS, and this may have a role in the blood pressure elevating effect of ginseng.

Concerning cinnamon, group V (diabetic treated with cinnamon) showed significant improvement in systolic blood pressure when compared with group IV (the diabetic untreated group). In diabetic untreated rats, systolic blood pressure falls as a result of decreased cardiac output, hypovolemia due to osmotic diuresis, impairment of sympathetic innervation of heart and vessels (Borges et al., 2006). Treatment with cinnamon is associated with improvement of diabetes and diabetic sequel hence the associated decrease in blood pressure improved. Noori et al. (2012) stated that constituents of cinnamon could provide better antioxidant activity in kidney, liver and heart tissues of rat against toxic assaults. Badalzadeh et al. (2014) reported that regular administration of cinnamon extract improves cardiac hemodynamics and performance.

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دراسة مقارنة للمستخلص المائي للجنسن والقرفة على ذكور الجرذان البيضاء البالغة المصابة بمرض البوال السكري

عادل شلبي – عبد الرحمن محمد عبد المطلب – أحمد محمد فرج القط

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خليفة البحث: مرض البوال السكري هو أحد أكثر الأمراض شعوبًا في المجتمعات المعاصرة على اختلاف مسويتها المادية والثقافية، ويتبين هذا السرور المزمن في كثير من الحالات إذا كان المرض مشاهدًا باعتقادات أخذ механизمة حماية التفاصيل المادية والثقافية، وبالتالي الإفصاح بأعراض مثل السكتة الدماغية وقصور الشرايين التاجية والقرفة وغيرها.

وللنباتات الجنسن مكانته طبية خاصة قدما وحديثا. وأوضح الباحث بعض الدراسات أن له تأثير مضاد للالتهابات ومضاد للسرطان، إلا أن استخدامه أوسع اجتذاباً هو إعداد أن مقوي عام لمضادات الإجهاد، ويعتبر نبات القرفة واحداً من النباتات الطبية المعروفة منذ القديم ويحتوي على بعض المركبات التي لها دور فعال في تنظيم نسب السكر والدهون بالدم دون آثار جانبية.

الهدف من البحث: يبين تأثير المستخلص المائي للجنسن والمستخلص المائي للقرفة على ذكور الفئران البيضاء المصاب بمرض البوال السكري من حيث نسب كل من السكر والهيموجلوبيين السكري والدهون والبروتين المتفاعل "سي" وضغط الدم الإفقي.

مواد وطريقة البحث: استخدم في هذا البحث ثماني فئران ذكوراً وأثناً من السلالات المحلية كنموذج للدراسة. وقد قسمت الفئران إلى عشر مجموعات متساوية: المجموعة الأولى (مجموعة ضابطة 1 غير مصابة بداء السكري). تم إعطاؤها ماء مغطر بالفم.


ADEL SHALABY et al.
انطلاق البحث: أسفر العلاج بالجنسنج عن تحسن البروتين الدهني عالي الكثافة والبروتين المتفاعل "سي" تحسناً ذو دالة إحصائية عند مقارنة المجموعة الخامسة المصابة بالسكر والتي تم علاجها بالجنسنج مقارنة بالمجموعة الضابطة الرابعة، كما أسفر العلاج بالقرفة عن تحسن كلاً من الكولسترول والدهون الثلاثية والبروتين المتفاعل "سي" وضغط الدم الإقlesiاسي في المجموعة السابعة المصابة بالسكر والتي تم علاجها بالقرفة تحسناً ذو دالة إحصائية عند مقارنتها بالمجموعة الضابطة الرابعة. كما كان من الواضح أن العلاج بالجنسنج أو القرفة كان حامياً ضد حدوث السكر وتبعته من ارتفاع الدهون والبروتين المتفاعل "سي" وانخفاض وزن الجسم وضغط الدم الإقlesiاسي في المجموعات التي سبق علاجها بالجنسنج أو القرفة أوكلاهما معاً عند مقارنتهم بالمجموعة الضابطة الرابعة.

الاستنتاج: تسبب المستخلص المائي لكل من الجنسنج والقرفة - وعلى وجه الخصوص القرفة - في تحسين بعض تبعات داء السكري كنسب الدهون والبروتين المتفاعل "سي"، وربما يرجع ذلك الى قدرتها على زيادة إفراز هورمون الإنسولين من البنكرياس وزيادة حساسية الجسم له، وأيضاً خفض معدل امتصاص الكربوهيدرات ومعدل البناء الحيوي للدهون في الكبد عن طريق تثبيط الإنزيمات المسؤولة عن ذلك، كما كان لكل من الجنسنج والقرفة تأثير حماية من إحداث السكر بالألوكسان وربما يعود ذلك الى التأثير المضاد للأكسدة وقدرتهم على تنظيف الأنسجة من الشوارد الحرة.