Al-Azhar Med. J. *Vol. 45(3), July 2016, 527-543*
DOI: 10.12816/0033121/ORG. No. 96281 ***Issues_title_English:*** 2016 Vol.45 Issue 3

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

By

Adel Shalaby, Abd El-Rahman M. Abd El-Mottaleb and Ahmad M. Farag Alkot

Medical Physiology Department, Al-Azhar University

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, TAGs, LDL and CRP, whereas body weight, systolic blood pressure and HDL were significantly reduced. Hepatocytes were markedly infiltrated with fat, and showed marked reduction in mitochondria 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 hyperlipedemia, 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, oligopep-tides and polyacetylenic alcohols ***(Qian et al., 2006).*** Ginseng treatment improves mitochondrial and hypotha-lamus-pituitary-adrenal axis function and increases anabolic hormone secretion ***(Jung et al., 2016).*** Anti-oxidative and anti-inflamma-tory 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 Pharma-ceuticals 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 chess cloth. 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).***

5. Ethyl Ether (Analar, Nile Pharmaceutical-Egypt): For anesthesia.

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)***
- ► Glysated 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).

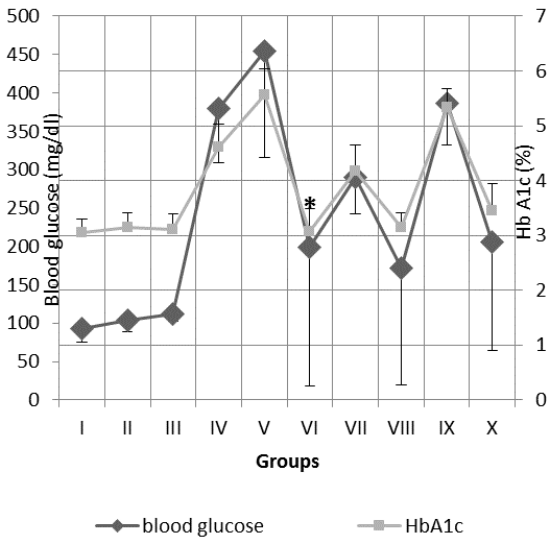

Figure (1): Concomitant levels of blood glucose and HbA1c levels in the studied groups (*): significant change when comparing with the diabetic non treated group IV

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).

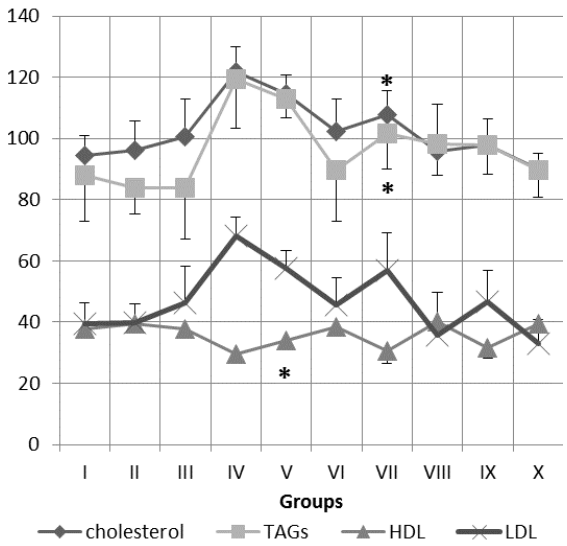

Figure (2): Concomitant levels of cholesterol, TAGs, HDL and LDL in studied groups (*): significant change when comparing with the diabetic non treated group IV

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).

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

(*): 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 anti-atherosclerotic 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 O^{-}_{2}, H_2O_2 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 mecha-nisms, 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 endocrinal 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 endocrinal 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.

REFERENCES

1. **Abbate S. L. and Brunzell J. D. (1990):** Pathophysiology of hyperlipedemia in diabetes mellitus. Journal of Cardiovascular Pharmacology, 16(9): s1-7.

2. **Ali M. M. and Agha F. G. (2009):** Amelioration of streptozotocin-induced diabetes mellitus, oxidative stress and dyslipidemia in rats by tomato extract lycopene. Scandinavian Journal of Clinical and Laboratory Investigation, 69(3): 371-379.

3. **Allain C. C., Poon L. S., Chan C. S. G., Richmond W. and Fu P. C. (1974):** Enzymatic determination of total serum cholesterol. Clinical Chemistry Journal, 20: 470-475.

4. **Askari, F., Rashidkhani B. and Hekmatdoost A. (2014):** Cinnamon may have therapeutic benefits on lipid profile, liver enzymes, insulin resistance, and high-sensitivity C-reactive protein in nonalcoholic fatty liver disease patients. Journal of Nutrition Research, 34 (2): 143-148.

5. **Azab K. Sh., Mostafa A. A., Ali E. M. M. and Abdel-Aziz M. A. S. (2011):** Cinnamon

extract ameliorates ionizing radiation-induced cellular injury in rats. Journal of Ecotoxicology and Environmental Safety, 74 (8): 2324-2329.

6. **Badalzadeh R., Shaghaghi M., Mohammadi M., Dehghan G. and Mohammadi Z. (2014):** The effect of cinnamon extract and long-term aerobic training on heart function, biochemical alterations and lipid profile following exhaustive exercise in male rats. Journal of Advanced Pharmaceutical Bulletin, 4 (2): 515-520.

7. **Bennion L. J. and Grundy S. M. (1977):** effects of diabetes mellitus on cholesterol metabolism in man, The New England Journal of Medicine, 296: 1365-1371.

8. **Borges G. R., de Oliveira M., Salgado H. C. and Fazan R. (2006):** Myocardial performance in conscious streptozotocin diabetic rats. Journal of Cardiovascular Diabetology, 5: 26.

9. **Braham D. and Trinder P. (1972)**: Estimation of glucose by glucose oxidase method. Journal of Analyst, 97: 142-145.

10. **Bromme H. J., Weinanday R., Peschke D. and Peschke E. (2001):** Estimation of the frequency of redox cycling between alloxan and dialuric acid. Hormone and Metabolism Research Journal, 33: 106-109.

11. **Brownlee M. (2005):** The pathobiology of diabetic complications. Journal of Diabetes, 54: 1618-1625.

12. **Buettner C., Yeh G. Y., Phillips R. S., Mittleman M. A. and Kaptchuk T. J. (2006):** Systematic review of the effects of ginseng on cardiovascular risk factors. The Annals of Pharmacotherapy Journal, 39: 83-95.

13. **Candido R., Forbes J. M., Thomas M. C., Thallas V., Dean R. G., Burns W. C., Tikellis C., Ritchie R. H., Twigg S. M., Cooper M. E. and Burrell L. M. (2003):** A breaker of advanced glycation end products attenuates diabetes-induced myocardial structural changes. Circulation Research Journal, 92:785–792.

14. **Cao H., Gravesc D. J. and Anderson R. A. (2010):** Cinnamon extract regulates glucose transporter and insulin-signaling gene expression in mouse adipocytes. Phytomedicine, 17 (13): 1027-1032.

15. **Chang K. S. K. and Lund D. D. (1986):** Alterations in the baroreceptor reflex control of heart rate in streptozotocin diabetic rats. Journal of molecular and cellular cardiology, 18 (6): 617–624.

16. **Cho W. C. S. Cho W. S., Chung S. K. W., Albert W. N., Christopher L., Cheng H. K. and Kevin K. M. (2006):** Ginsenoside Re of Panax ginseng possesses significant antioxidant and antihyperlipidemic efficacies in streptozotocin-induced diabetic rats. European Journal of Pharmacology, 550 (1): 173-179.

17. **Connell J. M. C., Whitworth J. A., Davies D. L., Lever A. F., Richards A. M. and Fraser R. (1987):** Effects of ACTH and cortisol administration on blood pressure, electrolyte metabolism, atrial natriuretic peptide and renal function in normal man. Journal of Hypertension, 5 (4): 425-435.

18. **Cooke D. W. and Plotnick L. (2008):** Type 1 diabetes mellitus in pediatrics. Pediatrics in Review Journal, 29(11): 374-384.

19. **David K. and Traci P. (2003):** Panax ginseng. American Family Physician Journal; Complementary and Alternative Medicine, 68 (8): 1539-1542.

20. **Du X-L., Edelstein D., Rossetti L., Fantus I. G., Goldberg H., Ziyadeh F., Wu J. and Brownlee M. (2000):** Hyperglycemia-induced mitochondrial superoxide overproduction activates the hexosamine pathway and induces plasminogen activator inhibitor-1 expression by increasing Sp1 glycosylation. Proceedings of the National Academy of Sciences of the United States of America, 97: 12222–12226.

21. **Eleazer S. (2003):** Diabetes in animals: contribution to understanding diabetes by study of its etiopathology in animal model. In DM, 6th edition, Chap. 16, pp. 231-332. Pbl. Mc Graw- Hill Camp, USA.

22. **Elsner M., Guldbakke B., Tiedge. M., Munday R. and Lenzen S. (2002):** Relative importance of transport and alkylation for pancreatic beta-cell toxicity of streptozotocin. Journal of Diabetologia, 43: 1528-1533.

23. **Ene A. C., Nwankwo E. A. and Samdi L. M. (2007):** Alloxan-induced diabetes in rats and the effects of black caraway (Carum Carvi L.) oil on their body weight. Research Journal of Medicine and Medical Sciences, 2(2): 48-52.

24. **Esser N., Legrand-Poels S., Piette J., Scheen A. J. and Paquot N. (2014):** Inflammation as a link between obesity, metabolic syndrome and type 2 diabetes. Diabetes Resarch and Clinical Practice Journal, 105: 141–150.

25. **Fazan Jr R., da Silva V. J. D., Ballejo G. and Salgado H. C. (1999):** Power spectra of arterial pressure and heart rate in streptozotocin induced diabetes in rats. Journal of Hypertension, 17 (4): 489–495.

26. **Fossati P. and Prencipe L. (1982):** Serum triglycerides determined calorimetrically with an enzyme that produces hydrogen peroxide. Clinical Chemistry Journal, 28(10): 2077-2080.

27. **Frier B. M. and Fisher M. (2006):** Diabetes Mellitus. In: Davidson's Principles and Practice of medicine, 20th edition, Pbl. Churchill Livingstone Elsevier, London, UK, Chapter 21: pp 810-813.

28. **Friedewald W. T., Levy R. I. and Fredrickson D. S. (1972):** Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry Journal, 18: 499-502.

29. **Giacco F. and Brownlee M. (2010):** Oxidative stress and diabetic complications. Journal of Circulation Research, 107: 1524-1571.

30. **Goyal B. R., Mesariya .P, Goyal R. K. and Mehta A. A. (2008):** Effect of telmisartan on cardiovascular complications associated with streptozotocin diabetic rats. Molecular and Cellular Biochemistry Journal, 314: 123–131.

31. **Green K., Brand M. D. and Murphy M. P. (2004):** Prevention of mitochondrial oxidative damage as a therapeutic strategy in diabetes. Diabetes Journal, 53: S110–118.

32. **Groove T. H. (1979):** The effect of reagent pH on the determination of high density lipoprotein cholesterol by precipitation with sodium phosphotungstate-magnesium. Clinical Chemistry Journal, 25: 560-564.

33. **Gun-sub S., Ki-Seung S., Kyoung-Won L., Chang-Won C., Ok-Hwan L., Jin-Ha L. and Chan-Kyu H. (2015):** Effects of puffed red ginseng powder and drink on blood glucose and serum lipid profile in streptozotocin-induced diabetic rats. Journal of Korean Social Food Scientific Nutrition, 44 (10): 1415-1421.

34. **Gupta Y. K., Sharma M. and Chaudhary G. (2001):** Antiepileptic activity of Panax Ginseng against pentylenetetrazole induced kindling in rats. Indian Journal of Physiology and Pharmacology, 45(4): 502-506.

35. **Hafizur R. M., Hameed A., Shukrana M., Raza S. A., Chishti S., Kabir N. and Siddiqui R. A. (2015):** Cinnamic acid exerts anti-diabetic activity by improving glucose tolerance in vivo and by stimulating insulin secretion in vitro Phytomedicine, 22 (2): 297-300.

36. **Hartel C., Strunk T., Bucsky P. and Schultz C. (2004):** Effects of vitamin C on intracytoplasmic cytokine production in human whole blood monocytes and lymphocytes. Journal of Cytokine, 27: 101-106.

37. **Hu F. B., Meigs J. B., Li T. Y., Rifai N. and Manson J. A. E. (2004):** Inflammatory markers and risk of developing type 2 diabetes in women. *Diabetes* Journal, 53: 693-700.

38. **Jackson C. V. and Carrier G. O. (1983):** Influence of short-term experimental diabetes on blood pressure and heart rate in response to norepinephrine and angiotensin II in the conscious rat. Journal of Cardiovascular Pharmacology, 5 (2): 260-265.

39. **Jarvill-Taylor K. J. and Graves D. J. (2001):** A hydroxychalcone derived from cinnamon functions as a mimetic for insulin in 3T3-L1 adipocytes. Journal of the American College of Nutrition, 20: 327-36.

40. **Jeon W. J., Oh J. S., Park M. S. and Jil G. E. (2013):** Anti-hyperglycemic effect of fermented ginseng in type 2 diabetes mellitus mouse Model. Phytotherapy Research, 27 (2): 166-172.

41. **Jialal I., Devaraj S. and Venugopal S. K. (2004):** C-reactive protein: risk marker or mediator in atherothrombosis. Journal of Hypertension, 44: 6-11.

42. Joshua J., Wang X., Spanakis E., Seemand T., Wand G., Needham B. and Golden S. H. (2015): Diurnal salivary cortisol, glycemia and insulin resistance: The multi-ethnic study of atherosclerosis. Journal of Psychoneuroendocrinology, 62: 327-335.

43. Jung D-H., Lee Y-J., Kim C-B., Kim J-Y., Shine S-H. and Park J-K. (2016): Effects of ginseng on peripheral blood mitochondrial DNA copy number and hormones in men with metabolic syndrome: A randomized clinical and pilot study. Complementary Therapies in Medicine Journal, 24: 40-64.

44. Kaiser N., Sasson S., Feener E. P., Boukobza-Vardi N., Higashi S., Moller D. E., Davidheiser S., Przybylski R. J. and King G. L. (1993): Differential regulation of glucose transport and transporters of glucose in vascular endothelial and smooth muscle cells. Journal of Diabetes, 42: 80–89.

45. Kang K. S., Kim H. Y., Yamabe N., Nagai R. and Yokozawa T. (2006): Protective effect of sun ginseng against diabetic renal damage. Journal of Biological and Pharmaceutical Bulletin, 29 (8): 1678-1684.

46. Kannappan S., Jayaraman T., Rajasekar P., Ravichandran M. K. and Anuradha C. V. (2006): Cinnamon bark extract improves glucose metabolism and lipid profile in the fructose-fed rat Singapore Medical Journal, 47(10): 858-863.

47. Khan A., Bryden N. A., Polansky M. M. and Anderson R. A. (1990): Insulin potentiating factor and chromium content of selected foods and spices. Biological Trace Elements Research Journal, 24: 183-188.

48. Khan A., Safdar M., Khan. M. M. A, Khattak K. N. and Anderson R. A. (2003): Cinnamon improves glucose and lipids of people with type 2 diabetes. Journal of Diabetes Care, 26: 3215-3228.

49. Laakso M. (1996): Glycemic control and the risk for coronary heart disease in patients with non-insulin-dependent diabetes mellitus: The Finnish studies. Annals of Internal Medicine, 124(1- pt. 2): 127-130.

50. Lee A. Y. W. and Chung S. S. M. (1999): Contributions of polyol pathway to oxidative stress in diabetic cataract. Journal of Federation of the American Society of Experimental Biology, 13:23–30.

51. Lee J., Burkhart G. and Janssen A. (2004): Nuclear factor Kappa British Journal of Clinical Pharmacology, 38:981-993.

52. Liu Z., Li W., Li X., Zhang M., Chen L., Zheng Y., Sun G. Z., and Ruan C. C. (2013): Antidiabetic effects of malonyl ginsenosides from *Panax ginseng* on type 2 diabetic rats induced by high-fat diet and streptozotocin. Journal of Ethnopharmacology, 145 (1): 233-240.

53. Madkor H. R., Mansour S. W. and Ramadan G. (2011): Modulatory effects of garlic, ginger, turmeric and their mixture on hyperglycaemia, dyslipidaemia and oxidative stress in streptozotocin – nicotinamide diabetic rats. British Journal of Nutrition, 105: 1210-1217.

54. Mang B., Wolters M., Schmitt B., Kelb K., Lichtinghagen R., Stichtenoth D. O., and Hahn A. (2006): Effects of a cinnamon extract on plasma glucose, HbA1c and serum lipids in diabetes mellitus type 2. European Journal of Clinical Investigation, 36: 340-344.

55. Moon H. K., Kim K. S., Chung S. K. and Kim J. K. (2015): Effect of wild Korean ginseng (Panax ginseng) extract on blood glucose and serum lipid contents in rats with multiple low-dose streptozotocin-induced diabetes. Journal of Food Science and Biotechnology, 24 (4): 1505-1511.

56. Murthy H. N., Dandin V. S., Lee E. J. and Paek K. Y. (2014): Efficacy of ginseng adventitious root extract on hyperglycemia in streptozotocin-induced diabetic rats. Journal of Ethnopharmacology, 153 (3): 917–921.

57. Noori S., Azmat M. and Mahboob T. (2012): Study on antioxidant effects of cinnamon and garlic extract in liver, kidney and heart tissue of rat. Bioscience Research, 9(1): 17-22.

58. Nocerino E., Amato M. and Izzo A. A. (2000): The aphrodisiac and adaptogenic properties. Journal of Fitoterapia, 71: S1-S5.

59. Onderoglu S. Sozer S., Erbil K. M., Ortace R. and Lermioglu F. (1999): The evaluation of long term effects of cinnamon bark and

olive leaf on toxicity induced by streptozotocin administration to rats. Journal of Pharmaceutics and Pharmacology, 51: 1305-1312.

60. Panveloski-Costa A. C., Teixeira S. S., Ribeiro I. M. R., Serrano-Nascimento C., Neves R. X., R. R. Favaro, Seelaender M., Antunes V. R. and Nunes M. T. (2016): Thyroid hormone reduces inflammatory cytokines improving glycaemia control in alloxan-induced diabetic wistar rats. Acta Physiologica, 217(2): 130–140.

61. Ping H., Zhang G. and Ren G. (2010): Antidiabetic effects of cinnamon oil in diabetic KK-A*y* mice. Food and Chemical Toxicology, 48 (8): 2344-2349.

62. Qian T., Zhi-Hong Jiang Z. H. and Cia Z. (2006): High-performance liquid chromatography coupled with tandem mass spectrometry applied for metabolic study of ginsenoside Rb1 on rat. Analytical Biochemistry Journal, 352 (1): 87-96.

63. Rahman M. S., Asaduzzaman M., Munira S., Begum M. M., Rahman M. M., Hasan M., Khatun A., Maniruzzaman M., Islam M., Khan M. H. K., Rahman M., Karim M. R. and Islam M. A. (2016): Comparative study of anti-hyperglycemic and anti-hyperlipidemic effects of honey, Coccinia cordifolia and Hilsha fish oil in streptozotocin induced diabetic rats. Biology and Medicine (Aligarh) 8 (2): 272-279.

64. Saw C. L-L., Wu Q. and Kong A. N. T. (2010): Anti-cancer and potential chemo-preventive actions of ginseng by activating Nrf2 (NFE2L2) anti-oxidative stress/anti inflamatory pathways. Journal of Chinese Medicine, 5 (37): 1-7.

65. Schram M. T., Chaturvedi N., Schalkwijk C. G., Fuller J. H. and Stehouwer, C. D. (2005): Markers of inflammation are cross sectionally associated with microvascular complications and cardiovascular disease in type 1 diabetes. The Eurodiab prospective complications study. Diabetologia Journal, 48: 370-378.

66. Sedigheh A., Jamal M. S., Setorki M., Somayeh K., Mahmoud R. K., Azadeh A. and Shamsi F. (2011): Hypoglycaemic and hypolipidemic effects of pumpkin (*Cucurbita pepo* L.) on alloxan-induced diabetic rats. African Journal of Pharmacy and Pharmacology, 5(23): 2620-2626.

67. Shen Y., Fukushima M., Ito Y., Muraki E., Hosono T., Seki T. and Ariga T. (2010): Verification of the antidiabetic effects of cinnamon using insulin-uncontrolled type 1 diabetic rats and cultured adipocytes. Bioscience, Biotechnology, and Biochemistry Journal, 74 (12): 2418-2425.

68. Shen Y., Honma N., Kobayashi K.., Jia L. N., Hosno T., Shindo K., Ariga T. and Seki T. (2014): Cinnamon extract enhances glucose uptake in 3T3-L1 adipocytes and C2C12 myocytes by inducing LKB1-AMPactivated protein kinase signaling. PLoS ONE 9(2): e87894.

69. Shihabudeen H. M. S., Priscilla D. H. and Thirumurugan K. (2011): Cinnamon extract inhibits α-glucosidase activity and dampens postprandial glucose excursion in diabetic rats. Journal of Nutrition & Metabolism, 20118: 46.

70. Stanely P., Prince M. and Venugopal P. M. (2000): Hypoglycaemic and other related actions of Tinospora cordifolia roots in alloxan-induced diabetic rats. Journal of Ethnopharmacology, 70: 1-9.

71. Szkudelski T. (2001). The mechanism of alloxan and streptozotocin action in β cells of rat pancreas. Physiology Research Journal, 50: 536-546.

72. Tangvarasittichai S., Sanguanwong S., Sengsuk S. and Tangvarasittichai O. (2015): Effect of cinnamon supplementation on oxidative stress, inflammation and insulin resistance in patients with type 2 diabetes mellitus. International Journal of Toxicological and Pharmacological Research, 7 (4): 158-164.

73. Thomson M., Al-Amin Z. M., Al-Qattan K. K., Lamia H. Shaban L. H. and Ali M. (2007): Antidiabetic and hypolipidaemic properties of garlic (Allium sativum) in stereptozotocin induced diabetic rats. International Journal of Diabetes and Metabolism, 15:108-115.

74. Tousoulis D., Papageorgiou N., Androulakis E., Siasos G., Latsios G., Tentolouris K. and

Stefanadis C. (2013): Diabetes mellitus-associated vascular impairment; novel circulating biomarkers and therapeutic approaches. Journal of the American College of Cardiology, 62 (8): A22-A26.

75. **Urdal P., Borch S. M., and Landaas S. (1992):** Rapid immunometric measurement of C-reactive protein in whole blood. Clinical Chemistery Journal, 38(4): 580-584.

76. **Vafa M., Farhad M., Farzad S., Hossein M. S. S., Iraj H., Banafshe G. and Sadat F. A. (2012):** Effects of cinnamon consumption on glycemic status, lipid profile and body composition in type 2 diabetic patients. International Journal of Preventive Medicine, 3: 531-536.

77. **Wei-Yi O., Farooqui T., Hwee-Ling K. Akhlaq A. F. and Eng A. L. (2015):** Protective effects of ginseng on neurological disorders. Frontiers in Aging Neuroscience Journal, 7: 129-137.

78. **Whitworth J. A., Brown M. A., Kelly J. J. and Williamson P. M. (1995):** Mechanisms of cortisol-induced hypertension in humans. Journal of Steroids, 60 (1): 76-80.

79. **Xie W. and Du L. (2011):** Diabetes is an inflammatory disease: evidence from traditional Chinese medicine. Journal of Diabetes. Obesity and Metabolism, 13: 289-301.

80. **Yoshida T., Yamagishi S., Nakamura K., Matsui T., Imaizumi T. Takeuchi M., Koga H., Ueno T. and Sata M. (2006):** Telmisartan inhibits AGE-induced C-reactive protein production through downregulation of the receptor for AGE via peroxisome proliferator-activated receptor-gamma activation. Diabetologia, 49: 3094–3099.

81. **Zander R., Lang W. and Wolf H. U. (1984):** Alkaline haematin D-575, a new tool for the determination of hemoglobin as an alternative to the cyanhaemiglobin method, I Description of the method. Clinical Chemistry Act Journal, 136(1): 83-93.

دراسة مقارنة للمستخلص المائى للجنسنج والقرفة على ذكور الجرذان البيضاء البالغة المصابة بمرض البوال السكرى

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

قسم الفسيولوجيا الطبية – كلية طب الأزهر

خلفية البحث: مرض البوال السكري هو أحد أكثر الأمراض شيوعا في المجتمعات المعاصرة علي إختلاف مستوياتها المادية والثقافية، ويتسبب هذا المرض المزمن في كثير من المضاعفات إذ أنه يكون عادة مصحوبا بإرتفاع نسب الدهون في الدم مما يساعد علي تصلب الشرايين، وبالتالي الإصابة بأمراض مثل السكتة الدماغية وقصور الشرايين التاجية والقدم السكري وغيرها.

ولنبات الجنسنج مكانة طبية خاصة قديما وحديثا ،وأوضحت بعض الدراسات أن له تأثير مضاد للإلتهبات ومضاد للسرطان، إلا أن إستخدامه الأوسع إنتشارا هو بإعتبار أنه مقوي عام ومضاد للإجهاد. ويعتبر نبات القرفة واحداً من النباتات الطبية المعروفة منذ القدم ويحتوي علي بعض المركبات التي لها دور فعال في تنظيم نسب السكر والدهون بالدم دون آثار جانبية

الهدف من البحث: بيان تأثير المستخلص المائي للجنسنج والمستخلص المائي للقرفة علي ذكور الفئران البيضاء المصابة بمرض السكر من حيث نسب كل من السكر والهيموجلوبين السكري والدهون و البروتين المتفاعل "سي" و ضغط الدم الإنقباضي.

مواد وطرق البحث: إستُخدم في هذا البحث ثمانون فأراً ذكراً أبيضاً من السلالات المحلية كنموذج للدراسة. و قد قسمت الفئران الي عشر مجموعات متساوية: **المجموعة الأولى (مجموعة ضابطة 1غير مصابة بداء السكري):** تم إعطاؤها ماء مقطر بالفم.

المجموعة الثانية (مجموعة ضابطة 2 غير مصابة بداء السكري): تم إعطاؤها مستخلص الجنسنج بالفم بجرعة 100 مجم/كجم. **المجموعة الثالثة (مجموعة ضابطة 3 غير مصابة بداء السكري):** تم إعطاؤها المستخلص المائي للقرفة بالفم بجرعة 2 مللي/كجم. **المجموعة الرابعة (مجموعة ضابطة مصابة بالسكري 4):** تم إعطاؤها ماء مقطر بالفم. **المجموعة الخامسة (مجموعة مصابة بالسكري 5)**: تم إعطاؤها المستخلص المائي للجنسنج بالفم بجرعة 100 مجم/كجم. **المجموعة السادسة (مجموعة مصابة بالسكري 6):** تم إعطاؤها المستخلص المائي للجنسنج بالفم بجرعة 100 مجم/كجم يوميا طوال التجربة ثم تم إحداث السكر بها. **المجموعة السابعة (مجموعة مصابة بالسكري 7)**: تم إعطاؤها المستخلص المائي للقرفة بالفم بجرعة 2 مللي/فأر. **المجموعة الثامنة (مجموعة مصابة بالسكري 8)**: تم إعطاؤها المستخلص المائي للقرفة بالفم بجرعة 2 مللي/فأر يوميا طوال التجربة ثم تم إحداث السكر بها. **المجموعة التاسعة (مجموعة مصابة بالسكري 9)**: تم إعطاؤها المستخلص المائي للجنسنج بالفم بجرعة 100 مجم/كجم و المستخلص المائي للقرفة بالفم بجرعة 2 مللي/فأر. **المجموعة العاشرة (مجموعة مصابة بالسكري 10)**: تم إعطاؤها المستخلص المائي للجنسنج بالفم بجرعة 100 مجم/كجم و المستخلص المائي للقرفة بالفم بجرعة 2 مللي/فأر يوميا طوال التجربة ثم تم إحداث السكر بها.

وقد تم إحداث مرض السكر في كل من المجموعة الرابعة وحتي العاشرة عن طريق إعطاء مادة الألوكسان (160 ملجم/كجم) بالحقن في الغشاء البريتوني مسبوقة بجرعة من النيكوتيناميد (110مجم/كجم) تم حقنها في الغشاء البريتوني قبل الألوكسان بعشرين دقيقة وذلك لتقليل سمية الألوكسان. وقد إستمرت التجربة لمدة شهر، وفي نهاية التجربة تم تسجيل وزن الجسم وضغط الدم الإنقباضي ثم تم سحب عينات الدم وذلك لقياس نسبة السكر بالدم، نسبة الهيموجلوبين السكري،الكوليستيرول،الدهون الثلاثية، البروتين الدهني عالي الكثافة،البروتين الدهني منخفض الكثافة،وبروتين "سي" المتفاعل.

نتائج البحث: أسفر العلاج بالجنسنج عن تحسن البروتين الدهني عالي الكثافة والبروتين المتفاعل "سي" تحسنا ذو دلالة إحصائية عند مقارنة المجموعة الخامسة المصابة بالسكر والتي تم علاجها بالجنسنج مقارنة بالمجموعة الضابطة الرابعة، كما أسفر العلاج بالقرفة عن تحسن كلا من الكوليستيرول والدهون الثلاثية والبروتين المتفاعل "سي" وضغط الدم الإنقباضي في المجموعة السابعة المصابة بالسكر والتي تم علاجها بالقرفة تحسنا ذو دلالة إحصائية عند مقارنتها بالمجموعة الضابطة الرابعة. كما كان من الواضح أن العلاج بالجنسنج أو القرفة كان حاميا ضد حدوث السكر وتبعاته من إرتفاع الدهون والبروتين المتفاعل "سي" وانخفاض وزن الجسم وضغط الدم الإنقباضي في المجموعات التي سبق علاجها بالجنسنج أو القرفة أوكلاهما معا عند مقارنتهم بالمجموعة الضابطة الرابعة.

الإستنتاج: تسبب المستخلص المائي لكلٍ من الجنسنج والقرفة - وعلي وجه الخصوص القرفة - في تحسين بعض تبعات داء السكري كنسب الدهون والبروتين المتفاعل "سي"، وربما يرجع ذلك الي قدرتهما علي زيادة إفراز هرمون الانسولين من البنكرياس وزيادة حساسية الجسم له، وأيضا خفض معدل امتصاص الكربوهيدرات ومعدل البناء الحيوي للدهون في الكبد عن طريق تثبيط الإنزيمات المسئولة عن ذلك، كما كان لكل من الجنسنج والقرفة تأثير الحماية من إحداث السكر بالألوكسان وربما يعود ذلك الي التأثير المضاد للأكسدة وقدرتهما علي تنظيف الأنسجة من الشوارد الحرة.