Hypoglycemic, antihyperlipidemic and antioxidant effects of ginger and alpha-lipoic acid in experimentally diabetic rats

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ABSTRACT

Ginger and alpha-lipoic acid have recently gained attention as a potent antioxidant. The purpose of the present study was to investigate the possible therapeutic effects of daily oral administration of ginger and alpha-lipoic acid on diabetic rats. 42 Male albino rats (150-180gm) were divided into 7 groups; control, olive oil (vehicle control), alloxan induced diabetic (DM), ginger-treated (GN), alpha-lipoic acid -treated (ALA), diabetic rats treated with ginger (DM+GN) and diabetic rats treated with alpha-lipoic acid (DM+ALA). After 5 weeks of GN and ALA administration (50 and 30 mg/kg b. wt respectively), hematological, biochemical and histological parameters were investigated. Diabetic rats exhibited a significant decrease in erythrocytic parameters as well as significant increase of fasting blood sugar (FBS), glycated hemoglobin (HbA1c), total cholesterol (TCh), triglyceride (TG), low density lipoprotein (LDL), very low density lipoprotein (VLDL), and a decrease in the level of high density lipoprotein (HDL). In addition, diabetic rats showed a significant decrease in hepatic GSH and an increase in the content of malondialdehyde (MDA). The administration of GN and ALA caused a significant decrease in FBS, HbA1c, TCh, TG, and LDL- cholesterol, concomitant with a significant increase in HDL-cholesterol was recorded. Moreover, GN and ALA administration blunted the increase in MDA and stimulated the GSH production in the liver of diabetic rats. The findings of this study indicated that injection of GN and ALA corrected the erythrocytic parameters, lipid profile, blood sugar level in diabetic rats and showed antioxidative properties. Additionally, the therapeutic effect of GN and ALA was confirmed with histopathological examination of pancreas. It could be postulated that: (i) GN and ALA have protective and anti-hyperglycemic effects and (ii) overall anti-diabetic effects of ALA are better than those of GN, at least in this experimental condition.

Keyword: Ginger, Alpha-lipoic acid, Hypoglycemic, Lipid profile, antioxidants, alloxan, rats.

INTRODUCTION

Diabetes mellitus (DM) is a serious health problem which is responsible for many complications affecting various organs in the body and it is considered the third greatest cause of death all over the world (El-Hilaly et al., 2007). During the last twenty years, the prevalence of diabetes has increased dramatically in many parts of the world and the disease is now a worldwide public health problem (Pan et al., 2012). Despite remarkable progress in the management of diabetes mellitus by synthetic drugs, there has been a renewed interest in natural anti-diabetic agents, especially the medicinal plants that attributed with therapeutic virtues (Grover et al., 2002). High prevalence and long-term complications of diabetes mellitus have prompted a search for new oral hypoglycemic agents from such antidiabetic plants (Grover et al., 2002). Many plant extracts and plant products have been shown to have significant antioxidant activity as well as having hypoglycemic properties (Patel et al., 2012). Ginger (GN) is an underground rhizome of plant Zingiber officinale belonging to the family Zingibecaceae. It is considered now a common constituent of diet worldwide (Elsheater et al., 2009). It was reported that ginger has medicinal properties against digestive disorders, rheumatism, and diabetes (Afzal et al., 2001). In addition, phytochemical reports have shown that the main constituents of ginger are Gingerol, Shagaols, Zingerone and Paradol (Li et al. 2012). Gingerol related compounds from ginger has been identified as an antioxidant (Masuda et al., 2004) and α-ketoglutarate dehydrogenase. It is a natural compound which widely present in plants and animals with antioxidant properties (Ghibu et al., 2009). ALA improves insulin sensitivity and is found to be useful in the treatment of insulin resistance (Evans and Goldfine, 2000). The therapeutic actions of LA are based on unique antioxidant ALA/ dihydrolipoic acid (DHLA) system. This system is able to reduce not only reactive oxygen species (ROS) but also has the ability to reduce and regenerate many crucial antioxidants including vitamin C, vitamin E and glutathione for this reason it is called as an antioxidant of antioxidants (Bilska et al., 2008). In the present study, the hypoglycemic, hypolipidemic and antioxidat properties of ginger and ALA were assessed in an animal model of diabetes.

MATERIALS AND METHODS

Chemicals

Alloxan monohydrate was obtained from sigma chemicals company, Egypt. Ginger (GN) tablets were obtained from the Arab Company for Pharmaceuticals and Medicinal plants (MEPACO), Egypt. Alpha lipoic acid powder was obtained from EVA Medical Company, Egypt. Thiobarbituric acid aqueous solution (TBA), n-butanol, pyridine, 1,1,3,3-tetramethoxypropane standard, trichloroacetic acid (TCA), phosphate buffer, 5,5-dithiobis(2-nitrobenzoic acid) (DTNB), and reduced glutathione (GSH) standard were obtained from Fluka (Taufkirchen, Germany). All the chemicals used were analytical grade. The assay kits for cholesterol, triacylglycerol, low density lipoprotein

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cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) were obtained from BioDiagnostic Company, Giza, Egypt.

**Animals and care**

Forty two healthy adult male Sprague-Dawley rats (150 – 180 g) were obtained from the Animal Breeding House in Faculty of Veterinary Medicine, Suez Canal University, Ismailia, Egypt. They were acclimatized to the environment for a week prior to the start of the experiments. The animals were kept in plastic cages under 12 h light/dark cycles with 25 ±3 °C. They were maintained on standard pellet diet and tap water ad libitum. The experiments were performed in accordance with the internationally accepted standard ethical guidelines for laboratory animal use and care as described in the European Community guidelines (EC, 1986).

**Induction of diabetes**

Animals were fasted for 24 hours and injected with freshly prepared aqueous solution of alloxan monohydrate dissolved in saline (150 mg/kg, i.p.) as reported previously (Satyanarayana et al. 2001). After the injection, they had free access of food and water. Treated rats were allowed to drink 5% glucose solution after 6 hrs of alloxan injection to counter hypoglycemic shock. The diabetic state was assessed by measuring the non-fasting plasma glucose concentration 72 hrs after alloxan treatment using the AccuCheck device (Roche Diagnostics, Indianapolis, IN). Blood was collected from tail vein and rats with a blood glucose level above 250 mg/dl were selected for the study and considered as diabetics (Zhang et al., 2006).

**Experimental Protocol**

Animals were then randomly divided into seven groups consisting of 6 rats in each group; Group I (non-diabetic control receive saline); Group II (non-diabetic group receive olive oil; solvent of ALA); Group III (non-diabetic treated GN); Group IV(non-diabetic treated ALA); Group V (diabetic control); and Group VI (diabetic treated GN group); Group VII (diabetic treated ALA group). GN and ALA were given orally to the rats through a gastric tube daily for 5 weeks at a dose of 50 and 30 mg/kg b. wt, respectively.

**Collection of blood and tissue samples.**

At the end of the 5 weeks post-treatment, Animals were fasted overnight then Blood was withdrawn retro-orbitally from the inner canthus of the eye with the help of capillary tube under mild ether inhalation anesthesia (Fernández et al., 2010). Blood samples were collected in test tubes and allowed to clot for 10 min. Serum was separated by centrifuging the samples at 3000 rpm for 10 min and stored in a refrigerator until analyzed (at -20 °C) until analyses of certain biochemical parameters. The animals were sacrificed after blood collection by cervical dislocation. The pancreas was then quickly dissected out, washed in ice-cold saline and stored in 10% formalin for tissue characterization.

**Hematological and biochemical analysis**

Haematological parameters were evaluated by electronic haematological counter (selectra coulter, Germany). Blood glucose level was determined by using the AccuCheck device (Roche Diagnostics, Indianapolis, IN). Triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-Ch) and low-density lipoprotein cholesterol (LDL-Ch) were measured at the end of the treatment period. The concentrations of total cholesterol was determined using the method of Richmond (1973). HDL-cholesterol was measured by enzymatic colorimetric methods of Jacobs et al. (1990). Triglyceride was measured by the fossati method associated with trinder reaction (Fossati and Precipe, 1982), while very low density lipoprotein cholesterol (VLDL-Ch) was calculated as TG/5, and LDL-Ch was calculated using the formula of Friedewald et al. (1972), where LDL-Ch= TCh - [HDL-Ch + (TG/5)]. Liver tissues from different groups were minced and homogenized (10% w/v), separately, in icecold 1.15% KCl–0.01M sodium, potassium phosphate buffer (pH 7.4) with an electric homogenizer. The homogenate was centrifuged at 4,000g for 20min, and the resultant supernatant was used for oxidative stress parameters. The levels of reduced glutathione (GSH) were determined according to Ellman (1959). The levels of lipid peroxidation (LP) were determined by measuring the content of the thiobarbituric acid reactive substances (TBARS) in the tissue homogenates following the procedure of Hogberg et al. (1974). According to this method, TBARS assay values are usually reported in malonaldehyde (malondialdehyde, MDA) equivalents, a compound that results from the decomposition of polyunsaturated fatty acid lipid peroxides.

**Histopathological examination**

Pancreas samples were taken from rats in different treated groups, and then fixed in 10% formal saline solution for twenty four hours. Washing was done in tap water followed by serial dilutions of absolute ethyl alcohol for dehydration. Specimens were cleared in xylene and embedded in paraffin at 56 °C in a hot air oven for twenty four hours. Paraffin wax tissue blocks were prepared for sectioning at 4 microns thickness by microtome. The obtained tissue sections were stained by hematoxylin and eosin stain for histopathological examination (Banchroft et al. 1996).

**Statistical analysis**

The results were presented as the mean ± standard error (SE) for six animals in each group. Statistically significant differences between groups were calculated using one- way analysis of variance (ANOVA) followed by post hoc multiple comparison test (Duncan). The
criterion for significance was set at $p \leq 0.05$ (Field, 2000).

**RESULTS**

Normal levels of FBS and HbA1c in healthy adult rats were measured as 84.67±3.06 mg/dl and 3.00 ± 0.07, respectively. Alloxan treated rats showed a significant elevation in FBS and HbA1c levels with percent of change equal to 421.63 and 48.33 respectively as compared with control group. Administration of GN (50 mg/kg b.wt.) and ALA (30 mg/kg b.wt.) in alloxan treated rats caused significant reduction in blood glucose level ($p \leq 0.05$) as compared with diabetic control rats (Fig. 1, A). Also, they caused a significant reduction ($p<0.05$) in HbA1c as compared to diabetic rats (Fig. 1, B).

![Figure 1: Effect of Ginger and α-lipoic on (A) blood glucose and (B) glycated hemoglobin level in normal and alloxan induced diabetic rats. Values as mean ± SE. n=6. One Way ANOVA followed by Duncan multiple comparison tests. $^a$ $p<0.05$ when compared with normal control group. $^b$ $p<0.05$ when compared with diabetic (D) group.](image)

As shown in Table 1, there was no significant change in erythrocytic parameters in normal rats treated with either GN or ALA. In Alloxan-diabetic rats, there were a significant decrease in red blood cell count, Hb, PCV, MCV, MCH and MCHC. These parameters were however significantly reversed by administration of GN and ALA when compared to the untreated diabetic group. However, GN and ALA were not able to restore the level of MCH back to that obtained in control group.

![Table 1: Effect of Ginger and α-lipoic on erythrogram in alloxan diabetic rats](image)

<table>
<thead>
<tr>
<th>Groups</th>
<th>Red blood cell count ($10^9$/μL)</th>
<th>PCV %</th>
<th>Hb (g/dL)</th>
<th>MCV (fL)</th>
<th>MCH (Pg)</th>
<th>MCHC %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>8.55 ± 0.30</td>
<td>46.76 ± 2.22</td>
<td>14.13 ± 0.62</td>
<td>54.60 ± 1.90</td>
<td>16.50 ± 0.18</td>
<td>31.35 ± 0.35</td>
</tr>
<tr>
<td>Olive oil</td>
<td>9.10 ± 0.07</td>
<td>45.20 ± 0.18</td>
<td>14.03 ± 0.09</td>
<td>55.66 ± 0.52</td>
<td>16.43 ± 0.21</td>
<td>31.03 ± 0.12</td>
</tr>
<tr>
<td>GN</td>
<td>7.90 ± 0.14$^a$</td>
<td>44.30 ± 0.47</td>
<td>13.75 ± 0.41$^a$</td>
<td>56.10 ± 0.63$^a$</td>
<td>16.30 ± 0.41</td>
<td>31.6 ± 0.21$^a$</td>
</tr>
<tr>
<td>ALA</td>
<td>8.28 ± 0.33</td>
<td>43.50 ± 1.03</td>
<td>13.26 ± 0.43</td>
<td>54.86 ± 1.99</td>
<td>16.08 ± 0.32</td>
<td>31.50 ± 0.73</td>
</tr>
<tr>
<td>D</td>
<td>7.15 ± 0.24$^a$</td>
<td>40.50 ± 0.35$^a$</td>
<td>11.10 ± 0.32$^a$</td>
<td>49.60 ± 1.80$^a$</td>
<td>15.50 ± 0.19$^a$</td>
<td>27.40 ± 0.88$^a$</td>
</tr>
<tr>
<td>D+GN</td>
<td>8.61 ± 0.30$^b$</td>
<td>43.87 ± 1.69$^b$</td>
<td>13.53 ± 0.53$^b$</td>
<td>55.95 ± 0.86$^b$</td>
<td>15.97 ± 0.26</td>
<td>31.35 ± 0.37$^b$</td>
</tr>
<tr>
<td>D + ALA</td>
<td>8.45 ± 0.15$^b$</td>
<td>42.30 ± 1.26$^b$</td>
<td>13.00 ± 0.36$^b$</td>
<td>53.96 ± 0.70$^b$</td>
<td>16.00 ± 0.35</td>
<td>30.06 ± 0.41$^b$</td>
</tr>
</tbody>
</table>

Values as mean ± SE. n=6. One Way ANOVA followed by Duncan multiple comparison tests. $^a$ $p<0.05$ when compared with normal control group. $^b$ $p<0.05$ when compared with diabetic (D) group.
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Table (2): Effect of Ginger and α-lipoic on lipid profile in alloxan diabetic rats

<table>
<thead>
<tr>
<th>Groups</th>
<th>Total cholesterol (mg/dl)</th>
<th>HDL cholesterol (mg/dl)</th>
<th>LDL cholesterol (mg/dl)</th>
<th>Triglycerides (mg/dl)</th>
<th>VLDL (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>67.63 ± 2.72</td>
<td>37.70 ± 4.2</td>
<td>13.33 ± 0.42</td>
<td>49.80 ± 3.69</td>
<td>9.96 ± 0.73</td>
</tr>
<tr>
<td>Olive oil</td>
<td>61.30 ± 3.38</td>
<td>31.67 ± 1.29</td>
<td>10.67 ± 0.76</td>
<td>66.20 ± 9.67</td>
<td>13.24 ± 1.93</td>
</tr>
<tr>
<td>GN</td>
<td>76.95 ± 6.35</td>
<td>37.97 ± 2.60</td>
<td>13.17 ± 1.68</td>
<td>61.70 ± 7.20</td>
<td>12.34 ± 1.44</td>
</tr>
<tr>
<td>ALA</td>
<td>71.28 ± 8.03</td>
<td>36.40 ± 2.30</td>
<td>15.00 ± 2.63</td>
<td>46.31 ± 10.07</td>
<td>9.26 ± 2.01</td>
</tr>
<tr>
<td>D</td>
<td>104.00 ± 0.36*</td>
<td>30.00 ± 3.01*</td>
<td>24.67 ± 1.47*</td>
<td>117.00 ± 0.36*</td>
<td>23.40 ± 0.07*</td>
</tr>
<tr>
<td>D + GN</td>
<td>81.16 ± 10.42*</td>
<td>38.87 ± 4.28*</td>
<td>14.00 ± 1.69*</td>
<td>99.12 ± 23.90*</td>
<td>19.82 ± 4.27*</td>
</tr>
<tr>
<td>D + ALA</td>
<td>61.68 ± 6.50*</td>
<td>39.00 ± 3.01*</td>
<td>9.50 ± 0.96*</td>
<td>54.53 ± 5.40*</td>
<td>10.91 ± 1.08*</td>
</tr>
</tbody>
</table>

Values as mean ± SE, n=6. One Way ANOVA followed by Duncan multiple comparison tests. *p<0.05 when compared with normal control group, †p<0.05 when compared with diabetic (D) group.

ocrine (Figure 3A). Pancreatic tissue from GN and alpha-lipoic treated non diabetic rats displayed normal structure (Figure 3C and 3E, respectively). The alloxan diabetic rats showed atrophy in the island of Langerhans cells (Figure 3B). However, treatment of alloxan-diabetic rats with either GN or ALA displayed recovery in size of island of Langerhans (Figure3 D and F, respectively).

**Figure (2):** Effect of Ginger and αlipoic on liver glutathione and lipid peroxidation content in normal and alloxan induced diabetic rats. Values as mean ± SE, n=6, One Way ANOVA followed by Duncan multiple comparison tests. a p<0.05 when compared with normal control group, †p<0.05 when compared with diabetic (D) group.

**DISCUSSION**

In the present study alloxan diabetic rats receiving GN or ALA showed a significant reduction of their fasting blood glucose values in comparison to diabetic control rats. The results are in consistent with Sharma and Shukla, (1977) who reported a significant blood glucose lowering effect of ginger juice in diabetic and non-diabetic animals. The results are also in corroboration with Sharma et al. (1996); Ahmed and Sharma, (1997) who reported hypoglycemic effects of ginger extract in diabetic induced animals. In addition, Akhani et al., (2004) stated that ginger pretreatment inhibited the induced hyperglycemia and hyperinsulinemia. Recent phytochemical studies have shown that the hypoglycaemic effect of ginger is due to presence of tannins and polyphenols with anti-oxidant property (Ugwoke and Nzekwe, 2012).

Oral administration of ALA for a period of five weeks significantly reduced the FBG in the alloxan-diabetic rats. This gave an indication of possible effects on the blood glucose metabolic pathway. Most authors who have reported on glycemic control potential of ALA have linked it with its ability to recruit glucose transporter-4 to plasma membranes similar to the insulin-stimulated glucose uptake (Singh and Jialal, 2008; Morakinyo et al., 2013).

A number of studies had shown significantly increased glycosylated hemoglobin levels in diabetic rats than the normal controls as seen in this study (Moussa, 2008; Ojo et al., 2013). Glycosylation is the interaction of glucose with the N-terminal amino acid of hemoglobin, producing glycosylated hemoglobin. Normally about one third of hemoglobin is glycosylated while it rises to two or three folds in diabetic patients. It persists all through the life span of RBC. Hence, estimation of glycosylated hemoglobin is an important diagnostic tool to assess the long-term control of blood glucose (WHO, 1999). H$_2$O$_2$ is produced as ROS and causes the release of iron from HbA1c more readily than normal hemoglobin. The iron is responsible for the generation of free radicals, which degrades various cell constituents. They also interact with DNA and proteins and bring out detrimental changes in their structure and properties. HbA1C is also subjected to auto-oxidation.
generating further oxidative stress (Aparna et al., 2012). In the present study, administration of GN effectively reduced HbAlc levels in diabetic rats. These results are in corroboration with Khadem Ansari et al. (2008) who showed decreased HbAlc levels by administration of ginger extract to the diabetic animals and this may be due to the decreased level of blood glucose.

The present results in agreement with a number of studies who reported that the enhancement of haematological parameters like MCH, MCV PCV, red blood cell count and Hb after GN (Ademola et al., 2009; Haghhighi and Ruhani, 2013) and ALA treatment (Bouasl et al., 2014) showed that GN and ALA can be useful in the treatment of anaemia and other blood disorders. The interpretation of hematological parameters enhancement after treatment with natural remedies has been subjected to wide speculation by many investigators. Tahmasebpour et al., (2013) reported that the increase in RBC, PCV, and WBC counts following administration of the methanol extract of _T. orientale_ may signify the positive effects on the haemopoietic system of experimental rats and might be capable of improving the hematological abnormalities associated with pathophysiology of diabetes mellitus. Similarly, it can be suggested that GN and ALA has a stimulatory effect on the bone marrow, which is responsible for production of red blood cells.

The present results elucidated that the rise in blood glucose was accompanied with marked increase in total lipids, triglycerides, total cholesterol and LDL-Ch in diabetic rats. These data were confirmed with the results of Battell et al. (1998) and Abdel-Moneim et al. (2002) who reported that, marked elevation in serum triglycerides, cholesterol and LDL-cholesterol levels in diabetic animals. This due to decrease in lipoprotein lipase (LPL) activity resulted from insulin deficiency (Minnich and Zilversmit, 1989). Diabetic dislipidemia is due mainly to decreased removal of triglycerides into the fat depots and the increase in the plasma concentration of LDL-cholesterol (Tsutsumi et al., 1995). According to the present results, there was a significant decrease in the levels of triglycerides and cholesterol after the administration of GN and ALA in comparison with diabetic animals. Hypolipidaemic effect of ginger extract was also demonstrated in cholesterol-fed rabbits (Bhandari and Grover, 1998). This may be an indication of progressive metabolic stress and decreased antioxidant levels are the leading factor in the pathogenesis and complication of diabetes mellitus. The stability and capacity of the antioxidant status during chronic diabetes seriously influences the outcome of the long-term complications caused by oxidative stress (Sasvari and Nyakas, 2003).

In the present study the observed significant increase in liver MDA and a decrease of GSH content in diabetic rats could be due to destruction of pancreatic β cells by alloxan probably through the generation of oxygen free radicals (Yang et al., 2010). Ginger decreases MDA concentration in normal, alloxan-induced and insulin resistant diabetic rats (Ajith et al. 2008; Heeba and Abd Elghany, 2010; Oboh et al., 2010), as it acts as a scavenger of oxygen radicals and also acts as an antioxidant.

Ginger extract possesses antioxidative characteristic since it can scavenger superoxide anion and hydroxyl radicals (Krishnakantha and Lokesh, 1993). Malondialdehyde (MDA) is the end-product of lipid peroxidation and the production of this aldehyde is used as a biomarker to measure the level of oxidative stress in an organism (Oboh, et al. 2010). Increased oxidative stress and decreased antioxidant levels are the leading cause of diabetes and diabetic complications (Jain, et al. 1999).
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1996; Feillet-Coudray, et al. 1999). Literature has shown that Zingiber officinale exhibit anti-oxidant effects (Stoilova et al. 2007; Ghasezmazeh, et al. 2010; Heeba & Abd-Elghany, 2010) which can be classified as a source of natural or phytochemical antioxidants (Kikuzaki and Nakatani, 1993). Treatment with 30 mg/kg a-lipoic acid for 5 weeks prevented the increase of MDA and increase hepatic GSH in diabetic rats. Feng et al. (2013) verified that one of the mechanisms by which ALA exerts this protective effect is via decreasing oxidative stress.

The present work concluded that the effect of GN and ALA administration on alloxan-induced diabetic rat showed hypoglycemic and antioxidant effects by enhancing liver GSH and down regulating the malondialdehyde (MDA) level. Also GN and ALA reduced significantly the plasma total cholesterol with its LDL fraction. Therefore, it may be regarded as a useful therapy for hyperlipidemia and the lowering of circulating cholesterol and the increased in HDL cholesterol

REFERENCES


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التأثيرات المحفظة لنسبة السكر والدهون ومضادات الأكسدة للزنجبيل وحمض الفا. ليوبوك في الجرذان المصاب بذاء بالسكري المستحث بالالوكسان

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ظاهرة في الأونة الأخيرة الاهتمام بالزنجبيل وحمض الفا. ليوبوك - باعتبارها أحد مضادات الأكسدة القوية، والمرض من هذا البحث هو دراسة أثر تناول الزنجبيل وحمض الفا. ليوبوك عن طريق الفم يوميا على قياسات الكريات الحمراء، ومستوي السكر في الدم والدهون والكولسترول في الجرذان. تم تقسيم العينة إلى 7 مجموعات. (1) المجموعة الضامنة، و (2) المجموعة المعالجة بزيت الزيتون، و (3) المجموعة الألوكرزان الذي يسبب السكري و (4 و 5) المطالعات بالزنجبيل، و (6 و 7) المطالعات بالزنجبيل وحمض الفا. ليوبوك.

هاي 5 أساليب من المعالجة أظهرت الجرذان المصاب بذاء السكري حديث إخفاق كبير في قياسات الكريات الحمراء، فضلا عن زيادة كبيرة في مستوى السكر في الدم، والدهون والكولسترول الكلي، الدهون الثلاثية، البروتين الدهني، منخفض الكثافة، و البروتين الدهني ذو الكثافة المنخفضة جدا، وإنخفاض في مستوى البروتين الدهني على الكثافة. كما أظهرت الفعالية الدهنية ملحوظ مولح في مستوى الكولسترول في جرذان عناية. و (8) أظهرت المعالجة للزنجبيل، وحمض الفا. ليوبوك أن الفا. ليوبوك كان لها تأثير كبير بين أكسدة الدهون وحمض الفا. ليوبوك. أن الفا. ليوبوك كان لها تأثير كبير بين أكسدة الدهون وحمض الفا. ليوبوك. نتائج هذه الدراسة تشير إلى أن معالجة مرض السكري باستخدام الزنجبيل أو حمض الفا. ليوبوك لها تأثيرات محفظة لزيادة نسبة الجلوكوز والدهون بالدم، ولكنها أوضح هذه الدراسة أن فاعلية حامض الفا. ليوبوك أكثر من الزنجبيل في علاج مرض السكري.