Biological Effects of Dried and Extracted Goldenberry on Diabetic Rats
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ABSTRACT
The objective of this study is to investigate the biological effects of dried goldenberry and its extracted on diabetic rats. Biological assay was conducted on diabetic mal-rats. The results indicated that supplementation with extract or dried goldenberry showed improve in relative organ weights (liver, heart, kidney, spleen, lungs and pancreas) and a significant increase in weight gain, food intake and food efficiency ratio. Serum blood glucose was significantly reduced in diabetic groups treated with extract or dried goldenberry at the end of experiment. This decrease was ranged from 98.33 mg/dl (G4) to 113.67 mg/dl (G5) as compared to positive control (204.33 mg/dl). The group fed on ethanolic goldenberry extract 500 mg/kg b.wt (G4) reported highest decrease in total cholesterol, triglycerides and LDL-C by means 90.67, 97.33 and 28.2 mg/dl, respectively and highest increase in HDL-C by mean 43 mg/dl in the same group as compared to positive control. On other hand, no significant (P>0.01) difference was found in uric acid, urea and creatinine values between negative control group and all treated groups except urea value of group fed on 10% dried goldenberry (G5). While, group that feed on extract or dried goldenberry had lower (P≤0.01) ALT, AST and ALP activities than positive control rats than the value of negative control group (G1). So we found goldenberry could be has therapeutic effect for diabetes and considered as a new source of bioactive and functional food.

Keywords: Biological, ethanolic extract, goldenberry, diabetic rats.

INTRODUCTION
Goldenberry, also known as Cape gooseberry, is the fruit of the plant Physalis peruviana L. that belongs to Solanaceae family. Goldenberry is a fruit with 4 -10 g of weight, approximately 1.25 – 2.50 cm of diameter, orange yellow skin and juicy pulp containing numerous small yellowish seeds (Legge, 1974 and Fischer, 1995). Goldenberry for chemical composition and they found that it contains 85.9, 1.5, 0.5, 11, 0.4 and 0.7 g/100g raw matter for moisture, protein, fat, carbohydrate, fiber and ash, respectively and 49 cal/100g for energy (Osorio and Roldan, 2003). Ca, Mg, K, P, Fe, Zn, Cu and Mn of goldenberry were 582, 1365, 20473, 4110, 40.5, 15.84, 8.81 and 8.11ppm, respectively (Endes et al., 2016). The ripeness stage of Cape gooseberry is directly proportional to many phytochemicals as vitamin C and β-carotene contents. A large amount of raw material properties (origin, ripeness stage, growing conditions etc.) differences in analytical methods, thermal and non-thermal processing have an effect on the extractability of the phytochemicals but also on the decrease of compounds and antioxidant activity (Olivares, 2017). The antioxidant capacity of dried and fresh goldenberry and reported that results indicated that the fresh sample (47.152 l/mol TE g-1d.b.) had significantly (P ≤ 0.05) higher antioxidant capacity than the dried samples (Izhi, et al., 2014). The total phenolic compound in samples of goldenberry is varies from 0.06 to 0.74 mg gallic acid equivalent/100 g fruit. On otherhand, the presence of ascorbic acid and phenolics in goldenberry fruit might contribute to the high level of antioxidant capacity, (Bravo et al., 2015). Diabetes mellitus as a one of the most common chronic diseases in nearly all countries, and continue to increase in numbers and significance, as economic development and urbanization lead to changing lifestyles characterized by reduced physical activity and increased obesity. Estimates of the current and future burden of diabetes are important in order to allocate community and health resources, to emphasize the role of lifestyle, and encourage measures to counteract trends for increasing prevalence (Whiting et al., 2011). The increasing prevalence of diabetes and its impact on morbidity and mortality have become global problems. In the United Kingdom, the prevalence of type 2 diabetes more than doubled from 2.39% in the year 2000 to 5.32% in 2013. The management of type 2 diabetes and its related complications, including retinopathy, kidney dysfunction, neuropathy, and foot problems accounts for about 10% of the entire National Health Service (NHS) budget in the UK (Sharma et al., 2016). The prevalence rate of diabetes in Egypt in 2008 was 4.07%. It increased with age, to reach 19.8% among females aged 50-59 (Naglaa et al., 2010). The enzymes (α-amylase and α-glucosidase) are present in the intestinal villi and they are necessary for polysaccharide digestion because they participate in the cleavage of these compounds to give monosaccharides (glucose, fructose, galactose). Thus, inhibition of the enzymes is one of the different possible mechanisms of antidiabetic drugs because such inhibition slows carbohydrate absorption and decreases postprandial blood glucose levels in both normal and diabetic subjects (fauci et al., 2009). Oral administration of goldenberry extract at a dosage of 200 mg/kg b.w daily for 30 days to diabetic rats showed significant (p<0.05) reducing in serum glucose and improved insulin level as compared with diabetic group. Chronic inflammation in adipose tissue together with obesity induces insulin. Diabetic groups showed significant increase in food intake when compared with control group of rats whoever the oral supplementation of goldenberry extract at a dosage of 200 mg/kg b.w daily for 30 days to diabetic rats found similar levels to that of control group of rats (sathyadevi et al., 2014). The aim of the current study was to investigate the biological effect of extract or dried goldenberry on diabetic rats.

MATERIALS AND METHODS
Materials:
Goldenberry (Physalis peruviana L.) collected from local market of Kafr El-Sheikh Governorate, Egypt in April 2017.
Chemicals used include alloxan was obtained from Morgan Co. Cairo, Egypt. While, Chemical kits for determination of serum glucose, total cholesterol (TC),
triglyceride (TG), high density lipoprotein (LDL), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), creatinine, urea, uric acid, were purchased from El-Gomhoria Company for Chemicals and Drugs El-Amelia, Cairo, Egypt.

Adult male albino rats, Sprague Dawley strain, were obtained from Research Institute of Ophthalmology, Giza, Egypt.

Methods:

Investigated plant:
Preparation of dried goldenberry fruits powder:

Goldenberry were selected by the same size and ripening stage then carefully washed with tap water and dried in sunny oven at 50 ± 5 °C until arriving by the moisture in the final product to about 8% then minced in moulinex machine (Al Araby for Electronic Manufacture Company, Egypt) then were put in polyethylene bags and packed in cartoon boxes until used.

Preparation of dried goldenberry fruits ethanolic extract:

100 g of dried fruit powder were weighed and poured into 500 ml conical flask and soaked with 500 ml 80% in distilled water ethanol distilled water for two days with daily shaking and kept in refrigerator covered by a piece of aluminum foil. Filter papers were used to purify the ethanolic extract and then the remains were extracted by the same way twice. The filtrates that were taken from extractions were mixed, and the filtrate was centrifuged at 3000 rpm for 10 min, then the ethanol was evaporated using a rotary evaporator apparatus attached with vacuum pump at 40 °C to obtain the powder. The obtained powder was weighted to determine the extracted yield then kept in light-protected containers at -25°C until further use.

Biological assay:

Animals and experimental design:

Thirty male albino rats (200g ±5) were housed individually in wire cages under the normal laboratory conditions. The rats were monitored for the external color, shape, appearance and distribution of hair and physical activity every day. The food and water were introduced to animals in special food cups to not loss or contamination. Food and water provided were reviewed every day. Rats weighted weekly. Rats were fed a standard diet (Ain, 1993) for 7 days as an adaptation period.

Experimental design:

Rats were randomly divided into six groups (n=5) and one of them was kept as normal control group. Rats were injected with alloxan (125 mg/kg body weight) according to the method described by (Desai and Bhide, 1985). The groups show as follows:
• Group 1 (-ve): normal rats fed on basal diet.
• Group 2 (+ve): diabetic rats fed on basal diet.
• Group 3: diabetic rats fed on basal diet and received ethanolic goldenberry extract, orally, in a dose of 250 mg/kg B.Wt.
• Group 4: diabetic rats fed on basal diet and received ethanolic goldenberry extract, orally, in a dose of 500 mg/kg B.Wt.
• Group 5: diabetic rats fed on basal diet + 10% dried goldenberry.
• Group 6: diabetic rats fed on basal diet + 15% dried goldenberry.

After the end of 28 days, blood samples were taken from rats. The blood samples were collected after 12 hours fasting from scarified rats then, put into dry clean centrifuge tubes. The blood was centrifuged for 10 minutes at 3500 rpm to separate the serum, which was carefully use pirated and transferred into clean quite plastic tubes and kept frozen at -18 °C until biochemical analysis (Malhotra, 2003).

Liver, heart, lungs, kidney, pancreas and spleen were removed and washed in saline solution, weighted and kept in formalin solution (10%, v/v) according to methods described by (Drury and Wallington, 1980).

The body weight was listed weekly during the period of experiment to determined body weight gain (BWG), feed intake by daily consumption of diet, feed efficiency ratio (FER) and relative organs weight according to (Chapman, et al., 1959) as follow:
• BWG (g) = Final Weight (g) – Initial Weight (g).
• BWG (%) = (BWG (g) ×100) / Initial Weight (g).
• FER = Gain in Body Weight (g) / Feed Intake (g).
• Relative Organs Weight = (Organ Weight (g) / Animal Body Weight (g) ×100).

Biological analysis:

Blood Glucose:

Glucose was determined by enzymatic test using chemical kits according to Trinder (1969).

Lipid profile:

TG (Triglycerides) was determined according to Fassati and Prencipe (1982). TC (Total Cholesterol) and HDL (high density lipoprotein) were determined according to description of (Allain, 1974). VLDL (Very low density lipoprotein) and LDL (low density lipoprotein) were carried according to (Lee and Nieman, 1996) and

Kidney functions:

Uric acid, urea and creatinine were analyzed according to the method of While et al. (1970); Malhotra (2003) and Henry (1974), respectively.

Liver functions:

ALT (Alanine Aminotransferase), AST (Aspartate Aminotransferase) and ALP (Alkaline Phosphatase) were determined according to Tietz (1976), Henry (1974) and Moss (1982), respectively.

Statistical Analysis:

Data was carried out using a completely randomized design (SAS, 1985) when there were significant major differences, the student-Newman-Keuls test was used to show the means. Differences among treatments of (P<0.01) were referred to significant changes.

RESULTS AND DISCUSSION

Effects of extract or dried goldenberry on body weight gain (g) and feed efficiency ratio of normal and diabetic rats:

Data reported in Table (1) showed the effects of extract or dried goldenberry on initial weight, final weight, BWG (g), BWG (%), feed intake and FER of normal and diabetic rats. The results indicated that no significant (P>0.01) difference in initial weight between all groups in the primary of experiment and ranged between 195g and 203.67g. At the end of experiment, the +ve group (G2) had lower (P≤0.01) final body weight, BWG (g), BWG (%), feed intake and FER than -ve group (G1).
This may be due to alloxan that associated with loss of body weight characteristics, which is due to increase muscle wasting and due to loss of tissue proteins (Kato et al., 2008).

Table 1. Effects of extract or dried goldenberry on BWG (g) and FER of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Groups Parameters</th>
<th>G1 (-ve)</th>
<th>G1 (+ve)</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial weight (g)</td>
<td>200.33±2.08</td>
<td>195±2.65</td>
<td>200.67±1.53</td>
<td>202.33±3.51</td>
<td>203.67±3.21</td>
<td>201.33±2.31</td>
<td>6.57</td>
</tr>
<tr>
<td>Final weight (g)</td>
<td>252.67±2.08</td>
<td>215±2.65</td>
<td>238±1</td>
<td>245.67±4.51</td>
<td>239±0</td>
<td>241±1</td>
<td>5.36</td>
</tr>
<tr>
<td>body weight gain (g)</td>
<td>52.33±2.08</td>
<td>20±2</td>
<td>37.33±2.08</td>
<td>43.33±4.04</td>
<td>35.33±3.21</td>
<td>39.67±2.52</td>
<td>6.88</td>
</tr>
<tr>
<td>body weight gain (%)</td>
<td>26.13±1.2</td>
<td>10.27±1.16</td>
<td>18.61±1.16</td>
<td>21.43±1.24</td>
<td>17.3±1.87</td>
<td>19.71±1.45</td>
<td>3.86</td>
</tr>
<tr>
<td>Feed intake (g)</td>
<td>18.5±0.75</td>
<td>14.87±1.0</td>
<td>15.27±0.64</td>
<td>17.23±0.71</td>
<td>15.03±1.02</td>
<td>16.8±0.96</td>
<td>2.15</td>
</tr>
<tr>
<td>feed efficiency ratio (FER)</td>
<td>2.83±0.13</td>
<td>1.35±0.11</td>
<td>2.45±0.17</td>
<td>2.51±0.21</td>
<td>2.35±0.08</td>
<td>2.49±0.29</td>
<td>0.44</td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤0.01).
G1 = -ve group. G2 = +ve group. G3 = fed on 250 mg/kg b.wt of goldenberry extract. G4 = fed on 500 mg/kg b.wt of goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

Effects of extract or dried goldenberry on organ weight of normal and diabetic rats:

Table 2. Effects of extract or dried goldenberry on liver, kidney, pancreas, lungs, heart and spleen weights (g) and relative weights of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Rats groups</th>
<th>Final Body Weight (g)</th>
<th>Liver Weight (g)</th>
<th>Relative Weight (%)</th>
<th>Kidney Weight (g)</th>
<th>Relative Weight (%)</th>
<th>Pancreas Weight (g)</th>
<th>Relative Weight (%)</th>
<th>Lungs Weight (g)</th>
<th>Relative Weight (%)</th>
<th>Heart Weight (g)</th>
<th>Relative Weight (%)</th>
<th>Spleen Weight (g)</th>
<th>Relative Weight (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>252.67±2.08</td>
<td>5.5±0.1</td>
<td>2.18±0.05</td>
<td>1.6±0.1</td>
<td>0.63±0.1</td>
<td>1.1±0.1</td>
<td>0.44±0.05</td>
<td>1.4±0.1</td>
<td>0.55±0.1</td>
<td>0.57±0.1</td>
<td>0.23±0.1</td>
<td>0.5±0.1</td>
<td>0.2±0.1</td>
</tr>
<tr>
<td>G2</td>
<td>215±1</td>
<td>6.3±0.1</td>
<td>2.93±0.05</td>
<td>1.7±0.05</td>
<td>0.79±0.1</td>
<td>0.93±0.1</td>
<td>0.44±0.05</td>
<td>1.54±0.14</td>
<td>0.72±0.1</td>
<td>0.87±0.1</td>
<td>0.4±0.1</td>
<td>0.28±0.1</td>
<td></td>
</tr>
<tr>
<td>G3</td>
<td>238±1</td>
<td>6.4±0.1</td>
<td>2.52±0.06</td>
<td>1.7±0.06</td>
<td>0.71±0.1</td>
<td>1.0±0.06</td>
<td>0.42±0.06</td>
<td>1.33±0.07</td>
<td>0.59±0.03</td>
<td>0.79±0.04</td>
<td>0.33±0.04</td>
<td>0.43±0.04</td>
<td>0.18±0.04</td>
</tr>
<tr>
<td>G4</td>
<td>245.67±4.51</td>
<td>5.7±0.1</td>
<td>2.28±0.04</td>
<td>1.67±0.06</td>
<td>0.68±0.06</td>
<td>1.17±0.04</td>
<td>0.48±0.04</td>
<td>1.58±0.04</td>
<td>0.64±0.04</td>
<td>0.79±0.04</td>
<td>0.32±0.04</td>
<td>0.5±0.02</td>
<td>0.2±0.04</td>
</tr>
<tr>
<td>G5</td>
<td>239±0</td>
<td>5.8±0.1</td>
<td>2.42±0.08</td>
<td>1.7±0.08</td>
<td>0.71±0.1</td>
<td>1.27±0.06</td>
<td>0.53±0.06</td>
<td>1.62±0.12</td>
<td>0.69±0.12</td>
<td>0.8±0.03</td>
<td>0.33±0.03</td>
<td>0.53±0.03</td>
<td>0.22±0.04</td>
</tr>
<tr>
<td>G6</td>
<td>241±1</td>
<td>5.3±0.1</td>
<td>2.21±0.03</td>
<td>1.6±0.03</td>
<td>0.66±0.06</td>
<td>1.17ab±0.49</td>
<td>1.35±0.56</td>
<td>0.77±0.32</td>
<td>0.43±0.18</td>
<td>0.43±0.18</td>
<td>0.32±0.04</td>
<td>0.43±0.18</td>
<td>0.18±0.04</td>
</tr>
<tr>
<td>LSD</td>
<td>5.36±0.51</td>
<td>0.22±0.02</td>
<td>0.27±0.11</td>
<td>0.11±0.09</td>
<td>0.19±0.08</td>
<td>0.08±0.17</td>
<td>0.07±0.09</td>
<td>0.31±0.13</td>
<td>0.29±0.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤0.01).
G1 = -ve group. G2 = +ve group. G3 = fed on 250 mg/kg b.wt of goldenberry extract. G4 = fed on 500 mg/kg b.wt of goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

Effects of extract or dried goldenberry on serum blood glucose of normal and diabetic rats:

The effects of extract or dried goldenberry on serum blood glucose of normal and diabetic rats are presented in Table (3). It can be notice that no significant (P>0.01) differences were found among all experimental rats before injury with diabetes. On other hand, as expected after injury with diabetes, the Serum glucose was significantly increasing except -ve group (G1).

While after 2 weeks, +ve group (204.73 mg/dl) still significantly higher than -ve group (91.67 mg/dl). All treated groups still significantly high as compared -ve group. These results in agreed with Mora et al. (2010) whose found that the oral administration of Physalis peruviana extract during 15 days reduced the blood glucose levels, further than 30%.

While after 4 weeks, positive control group (204.33 mg/dl) had a higher (P≤0.01) serum glucose than negative control group (91.33 mg/dl) and other treated rats.
Serum glucose was significantly reduced by the supplementation with goldenberry and its ethanolic extracts. The best reducing was for group that supplemented with ethanolic goldenberry extract 500 mg/kg b.wt (G4) (98.33 mg/dl) followed by group that supplemented with dried goldenberry 15% (G6) (101 mg/dl) then group that supplemented with ethanolic goldenberry extract 250 mg/kg b.wt (G3) (102.33 mg/dl). The results of Sathyadevi et al. (2014) support our findings.

Table 3. Effects of extract or dried goldenberry on serum blood glucose of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>G1 (-ve)</th>
<th>G2 (+ve)</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before injury</td>
<td>with diabetes mg/dl</td>
<td>86.72±1.96</td>
<td>88.32±1.55</td>
<td>87.77±1.31</td>
<td>86.27±1.45</td>
<td>88.1±0.87</td>
<td>88.67±</td>
<td>3.59</td>
</tr>
<tr>
<td>After injury</td>
<td>with diabetes mg/dl</td>
<td>86.76±2.33</td>
<td>200.7±2.14</td>
<td>198.63±1.25</td>
<td>202.7±1.4</td>
<td>199.93±1</td>
<td>203.1±0.36</td>
<td>3.9</td>
</tr>
<tr>
<td>After 2 weeks</td>
<td>mg/dl</td>
<td>91.67±0.78</td>
<td>204.73±1.82</td>
<td>122.97±2.25</td>
<td>119.93±1.18</td>
<td>134.3±2.15</td>
<td>121.67±0.84</td>
<td>4.04</td>
</tr>
<tr>
<td>After 4 weeks</td>
<td>mg/dl</td>
<td>93.1±4.04</td>
<td>204.33±4.51</td>
<td>102.33±2.89</td>
<td>98.33±1.53</td>
<td>113.67±1.53</td>
<td>101±1</td>
<td>7.25</td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤0.01).

G1 = -ve group. G2 = +ve group. G3 = feed on 250 mg/kg b.wt of ethanolic goldenberry extract. G4 = feed on 500 mg/kg b.wt of ethanolic goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

Effects of extract or dried goldenberry on lipids profile of normal and diabetic rats:

Elevated serum triglycerides (TG) levels were reviewed as a dependent risk factor in coronary heart disease and atherosclerotic cardiovascular disease (Pacheco et al., 2001). Data in Table (4) showed that the levels of TG (Triglycerides), TC (Total Cholesterol), VLDL (Very low density lipoprotein), LDL (low density lipoprotein) and HDL (high density lipoprotein) on -ve and diabetic groups supplemented with extract or dried goldenberry. Total cholesterol, triglycerides and VLDL levels in blood serum showed significant differences (P<0.01) between +ve groups (121.67, 137.67 and 27.53mg/dl, respectively) and -ve groups (83.33, 86 and 17.2 mg/dl, respectively) while group fed on ethanolic goldenberry extract 500 mg/kg b.wt (G4) were the nearest group to +ve group. Moreover, total cholesterol level of rats fed on ethanolic goldenberry extract 250 mg/kg b.wt (G3) did not record significant (P>0.01) differences with rats fed on 10% and 15% dried goldenberry. On other hand, triglycerides and VLDL levels of the same group (G3) showed significant differences (P<0.01) with rats fed on ethanolic goldenberry extract 500 mg/kg b.wt (G4) and 15% dried goldenberry (G5) and non-significant (P>0.01) differences with rats fed on 15% dried goldenberry (G6). Also, this value is still higher than this value of -ve group.

Data in the same Table presented that HDL level of -ve group had (42.67 mg/dl) which was significantly higher than +ve group (26 mg/dl) furthermore, did not show significant (P>0.01) differences with rats fed on ethanolic goldenberry extracts (250 – 500 mg/kg b.wt) and 15% dried goldenberry. On other hand, rats fed on 15% dried goldenberry showed non-significant (P>0.01) differences with +ve group.

Results displayed that LDL level of +ve group (68.13 mg/dl) was higher as compared with -ve group (27.13 mg/dl). Supplementation rats with 15% dried goldenberry (G6) and its ethanolic extract 500mg/kg b.wt (G4) appeared non-significant (P>0.01) differences with -ve rats. So it can be notice that supplementation rats with dried goldenberry and its ethanolic extracts significantly (P<0.01) improved HDL and reduced total cholesterol, total triglyceride , LDL and VLDL as compared with +ve rats.

Table 4. Effects of extract or dried goldenberry on lipids profile of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>G1 (-ve)</th>
<th>G2 (+ve)</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mg/dl)</td>
<td></td>
<td>83.33±4.04</td>
<td>121.67±4.51</td>
<td>97.33±3.289</td>
<td>90.67±1.53</td>
<td>101.33±1.53</td>
<td>95±1</td>
<td>4.03</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td></td>
<td>86±3.46</td>
<td>137.67±2.51</td>
<td>108.33±3.51</td>
<td>97.33±0.58</td>
<td>115.67±1.15</td>
<td>103.33±1.53</td>
<td>5.99</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td></td>
<td>42.67±4.62</td>
<td>26±3.61</td>
<td>35.33±2.31</td>
<td>43±3.61</td>
<td>31±1</td>
<td>39.67±0.58</td>
<td>7.48</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td></td>
<td>27.13±4.9</td>
<td>68.13±2.8</td>
<td>40.4±2.08</td>
<td>28.2±4.13</td>
<td>47.2±0.35</td>
<td>34.67±0.83</td>
<td>7.49</td>
</tr>
<tr>
<td>VLDL (mg/dl)</td>
<td></td>
<td>17.2±0.69</td>
<td>27.53±0.5</td>
<td>21.67±0.7</td>
<td>19.47±0.12</td>
<td>23.13±0.23</td>
<td>20.67±0.31</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤0.01).

G1 = -ve group. G2 = +ve group. G3 = feed on 250 mg/kg b.wt of ethanolic goldenberry extract. G4 = feed on 500 mg/kg b.wt of ethanolic goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

Hyperlipidemia is a complication associated with diabetes mellitus due to qualitative and quantitative abnormalities in lipoproteins (Miller et al., 2002). Chronic hyperglycemia in diabetes leads to over production of free radicals and these contribute to the development of diabetic nephropathy (Sharma et al., 2006).

Effects of extract or dried goldenberry on liver function:

Data in Table (5) showed the effects of extract or dried goldenberry on AST, ALT and ALP in normal and diabetic rats. -ve group had lower (P≤0.01) ALT, AST and ALP activities than +ve group. The increase level of serum transaminase in diabetic groups due to the enzymes were activated in the absence or shortage of insulin and also of increased availability of amino acids in diabetes, consequent to be responsible for the increased gluconeogenesis and ketogenesis observed in diabetes (Felg et al.,1970).

Rats that supplemented with dried goldenberry and its ethanolic extracts had lower (P≤0.01) ALT, AST and ALP activities than +ve group by different rates. Furthermore, rats fed on ethanolic goldenberry extract 500 mg/kg b.wt (G4) was more effective (P≤0.01) in reducing AST, ALT and ALP activities but still higher than the value of -ve group.
Moreover, AST and ALP values of rats fed on 15% dried goldenberry (G6) was lower than rats fed on ethanolic goldenberry extract 250 mg/kg b.wt (G3) but did not show significant (P=0.01) change.

On other hand, all values of ALP of all treated rats recorded significant (P<0.01) different as comparing with +ve group. No significant (P>0.01) difference was found in AST/ALT values between all experimental rats. These results were in the same line with Sathyadevi et al. (2014) whose found that the activities of serum AST, ALT and ALP in diabetic rats showed significant (p < 0.05) increasing as compared with control group. Oral supplemented of goldenberry extract to diabetic rats significantly (p < 0.05) normalized the differed levels in comparison with control group.

Table 5. Effects of extract or dried goldenberry on liver function (AST, ALT, AST/ALT and ALP) of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Groups Parameters</th>
<th>G1 (-ve)</th>
<th>G1 (+ve)</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST U/L</td>
<td>99.3± 7.45</td>
<td>134.67± 0.55</td>
<td>116.33± 1.53</td>
<td>101± 4.36</td>
<td>120.23± 0.9</td>
<td>114.67± 1.53</td>
<td>9.12</td>
</tr>
<tr>
<td>ALT U/L</td>
<td>43.67± 1.27</td>
<td>62.5± 1.47</td>
<td>52.2± 1.13</td>
<td>47.43± 1.42</td>
<td>57.73± 0.51</td>
<td>50.6± 0.36</td>
<td>2.78</td>
</tr>
<tr>
<td>AST/ALT U/L</td>
<td>2.27± 0.11</td>
<td>2.15± 0.06</td>
<td>2.23± 0.05</td>
<td>2.13± 0.14</td>
<td>2.08± 0.02</td>
<td>2.27± 0.01</td>
<td>0.2</td>
</tr>
<tr>
<td>ALP U/L</td>
<td>175.67± 1.56</td>
<td>329.43± 1.62</td>
<td>200.77± 0.5</td>
<td>182.33± 6.03</td>
<td>209.77± 1.27</td>
<td>192.57± 1.1</td>
<td>6.79</td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤ 0.01).

G1= Nve group. G2 = +ve group. G3 = feed on 250 mg/kg b.wt of ethanolic goldenberry extract. G4 = feed on 500 mg/kg b.wt of ethanolic goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

Effects of extract or dried goldenberry on kidney function:

Urea and uric acid synthesized from ammonia produced as a result of the de-amination of amino acids in the liver, they consider the principal waste products of protein catabolism. Having high protein diet or by increased endogenous catabolism due to starvation or tissue damage is accelerated the rate of production (Bequette and Sunny, 2005).

The effect of extract or dried goldenberry on uric acid, urea and creatinine of normal and diabetic rats are shown in Table (6). Positive control group had higher (p<0.01) uric acid, urea and creatinine than negative control group. Supplementation rats with extract or dried goldenberry led to reduce the values. Rats fed on ethanolic goldenberry extract 500 mg/kg b.wt (G4) had the best reducing followed by rats fed on 15% dried goldenberry (G6) then rats fed on ethanolic goldenberry extract 250 mg/kg b.wt (G3) and rats fed on 10% dried goldenberry (G5) but No significant (P>0.01) difference was found in uric acid, urea and creatinine values between negative control group and all treated rats except urea value of rats fed on 10% dried goldenberry (G5).

Table 6. Effects of extract or dried goldenberry on uric acid, urea and creatinine of normal and diabetic rats:

<table>
<thead>
<tr>
<th>Groups Parameters</th>
<th>G1 (-ve)</th>
<th>G1 (+ve)</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>G6</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric acid (mg/dl)</td>
<td>1.03± 0.11</td>
<td>2.29± 0.52</td>
<td>1.43± 0.22</td>
<td>1.22± 0.3</td>
<td>1.66± 0.37</td>
<td>1.23± 0.26</td>
<td>0.8</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>26.33± 0.55</td>
<td>45.27± 1.91</td>
<td>28.3± 0.95</td>
<td>25.6± 1.21</td>
<td>31.23± 1.07</td>
<td>27.32± 1.23</td>
<td>3.06</td>
</tr>
<tr>
<td>creatinine (mg/dl)</td>
<td>0.24± 0.06</td>
<td>0.45± 0.06</td>
<td>0.32± 0.05</td>
<td>0.25± 0.06</td>
<td>0.38± 0.03</td>
<td>0.31± 0.03</td>
<td>0.13</td>
</tr>
</tbody>
</table>

Means ± standard deviations with different superscript letters in the same row are significantly different at (P≤ 0.01).

G1 = -ve group. G2 = +ve group. G3 = feed on 250 mg/kg b.wt of ethanolic goldenberry extract. G4 = feed on 500 mg/kg b.wt of ethanolic goldenberry extract. G5 = fed on 10% dried goldenberry. G6 = fed on 15% dried goldenberry. b.wt = body weight.

CONCLUSION

In our study the daily consumption of goldenberry reduced blood glucose on diabetic rats so our results suggest that the fruit of P. peruviana considered a potential agent for diabetes and considered as a new source of bioactive and functional food.

REFERENCES

تأتي البكتيريا البيولوجية للنوت الذهبي (المرجل) المحفوظ والمستخلص على الفترات المصابة بالسفر

1. مهندس بحث تكنولوجيا الأغذية، مركز البحوث الزراعية، جيزة، مصر
2. مهندس بحث تكنولوجيا الأغذية، جامعة المنصورة

نگلا، آ. و شفکی، A. و گهدا، E. (2010). The Epidemiology of Diabetes Mellitus in Egypt: Results of a National Survey. The Egyptian Journal of Community Medicine, 28: (3).