RESULTS
Despite advances in medicine, diabetes as a major health complication seems to be growing at an alarming proportion world over and in India. Traditional plant remedies have been used for centuries in the treatment of diabetes. Demand for plant products that are hypoglycemic agents is on the rise. Even though the bioactivity of various plant products is unknown, these are prescribed and used widely because of underlying faith in folk medicine based on time tested traditional practices.

Evaluated results of four such selected plants - *Melia composita* Willd., *Gmelina asiatica* Linn., *Sphaeranthus indicus* Linn., *Parmelia perlata* Ach. and polyherbal formulation prepared out of it on various biochemical and histological parameters in diabetic untreated and plant extract treated animals were presented in sequel.
## Results

### 6.1.1 Preliminary Phytochemical screening for plant extracts

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the Test</th>
<th>Reaction</th>
<th>MC</th>
<th>GA</th>
<th>SI</th>
<th>PP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Saponin – Water shake</td>
<td>Foam</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
<td>+ ve</td>
</tr>
<tr>
<td>2</td>
<td>Tannin – Lead acetate solution</td>
<td>White precipitate</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
</tr>
<tr>
<td>3</td>
<td>Sterol – Chloroform + acetic anhydride + H₂SO₄</td>
<td>Green colour</td>
<td>+ ve</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
</tr>
<tr>
<td>4</td>
<td>Terpenes – Tin + Thionyl chloride</td>
<td>Pink colour</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
</tr>
<tr>
<td>5</td>
<td>Flavonoids – Alcohol + Magnesium + drops of HCl</td>
<td>Magenta colour</td>
<td>- ve</td>
<td>+ ve</td>
<td>+ ve</td>
<td>- ve</td>
</tr>
<tr>
<td>6</td>
<td>Coumarins- 10% NaOH</td>
<td>Yellow colour</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
<td>- ve</td>
</tr>
<tr>
<td>7</td>
<td>Quinone – Conc H₂SO₄</td>
<td>Red Colour</td>
<td>- ve</td>
<td>+ ve</td>
<td>- ve</td>
<td>+ ve</td>
</tr>
<tr>
<td>8</td>
<td>Lignin – Alcoholic Phloroglucinol + Dil HCl</td>
<td>Red Colour</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
</tr>
<tr>
<td>9</td>
<td>Alkaloid – Drangendorff’s Reagent + acetic acid</td>
<td>Orange precipitate</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
</tr>
<tr>
<td>10</td>
<td>Starch – Iodine</td>
<td>Blue colour</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
</tr>
<tr>
<td>11</td>
<td>Gum – Drops of water</td>
<td>Swells</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
<td>- ve</td>
</tr>
<tr>
<td>12</td>
<td>Polyphenols – Folin’s reagent</td>
<td>Bluish green</td>
<td>+ ve</td>
<td>+ ve</td>
<td>+ ve</td>
<td>- ve</td>
</tr>
</tbody>
</table>

+ ve - indicates positive  
- ve - indicates negative

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation
Results

6.1.2.1 TLC Profile of Plant extracts

Sample: Plant extracts and PHF (1- PP, 2- GA, 3- MC, 4 - SI, 5 – PHF)

Mobile Phase: Methanol : water (8: 2)

Spraying Reagent: Folin’s phenol reagent

Table 2b

<table>
<thead>
<tr>
<th>S.No</th>
<th>Plant extract</th>
<th>Rf</th>
<th>Colour of the spot</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PP</td>
<td>0.82</td>
<td>blue</td>
</tr>
<tr>
<td>2</td>
<td>GA</td>
<td>0.83</td>
<td>blue</td>
</tr>
<tr>
<td>3</td>
<td>MC</td>
<td>0.82</td>
<td>blue</td>
</tr>
<tr>
<td>4</td>
<td>SI</td>
<td>0.83</td>
<td>blue</td>
</tr>
<tr>
<td>5</td>
<td>PHF</td>
<td>0.83</td>
<td>blue</td>
</tr>
</tbody>
</table>
6.1.2.2 TLC Profile of Plant extracts

Figure: 12

Sample: Plant extracts and PHF (1 - PP, 2 - GA, 3 - MC, 4 - SI, 5 - PHF)

Mobile Phase: Methanol : Acetic acid (10: 1)

Spraying Reagent: Iodine chamber

Table 2c

<table>
<thead>
<tr>
<th>S.No</th>
<th>Plant extract</th>
<th>Rf</th>
<th>Colour of the spot</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PP</td>
<td>0.81</td>
<td>Yellow</td>
</tr>
<tr>
<td>2</td>
<td>GA</td>
<td>0.74</td>
<td>Yellow</td>
</tr>
<tr>
<td>3</td>
<td>MC</td>
<td>0.70</td>
<td>Yellow</td>
</tr>
<tr>
<td>4</td>
<td>SI</td>
<td>0.72</td>
<td>Yellow</td>
</tr>
<tr>
<td>5</td>
<td>PHF</td>
<td>0.81</td>
<td>Yellow</td>
</tr>
</tbody>
</table>
### Table 3: Organic Analysis

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the phytoconstituents estimated</th>
<th>Sample Details</th>
<th>PP</th>
<th>GA</th>
<th>MC</th>
<th>SI</th>
<th>PHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Total Alkaloids (mg/Kg)</td>
<td></td>
<td>0.45</td>
<td>0.54</td>
<td>0.62</td>
<td>0.45</td>
<td>0.88</td>
</tr>
<tr>
<td>2.</td>
<td>Total Flavonoids (mg/Kg)</td>
<td></td>
<td>0.72</td>
<td>1.29</td>
<td>0.89</td>
<td>1.69</td>
<td>1.98</td>
</tr>
<tr>
<td>3.</td>
<td>Tannins (mg/Kg)</td>
<td></td>
<td>0.20</td>
<td>0.23</td>
<td>0.35</td>
<td>0.22</td>
<td>0.15</td>
</tr>
<tr>
<td>5.</td>
<td>Phenols (g/Kg)</td>
<td></td>
<td>1.0</td>
<td>1.6</td>
<td>1.2</td>
<td>2.0</td>
<td>2.13</td>
</tr>
</tbody>
</table>

Preliminary phytochemical screening of the plant extracts were carried out. It showed the presence of Coumarins, Tannins, Alkaloids and Phenols (Table 2a & 3). The presence of tannins and phenols was further confirmed by TLC profiles (Table 2b&2c, Figure – 11,12).

TLC profile of aqueous extract of the drugs on silica gel ‘G’ plate using methanol:water in the ratio of 8:2 as mobile phase and Folin’s phenol as spraying reagent revealed blue spots at Rf 0.82,0.83,0.82,0.83 & 0.83 (Table 2b, Figure – 11).

TLC profile of aqueous extract of the drugs on silica gel ‘G’ plate using methanol:acetic acid in the ratio of 10:1 as mobile phase. Yellow spots appeared on exposure to Iodine vapours at Rf 0.81,0.74,0.70,0.72 & 0.81 (Table 2c, Figure – 12).
6.2 TOXICITY EVALUATIONS – Short term toxicity study

Table -4 Toxicity evaluation of selected plant sources

<table>
<thead>
<tr>
<th>Groups/ Parameters</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>56.7±2.31</td>
<td>50.1±1.76</td>
<td>117.3±2.91</td>
</tr>
<tr>
<td><strong>MC</strong> Extract treated (mg/Kg b.w)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MC II (200)</td>
<td>52.1±1.81</td>
<td>48.6±3.10</td>
<td>112.3±2.71</td>
</tr>
<tr>
<td>MC III (300)</td>
<td>54.7±2.32</td>
<td>47.5±2.71</td>
<td>114.2±2.04</td>
</tr>
<tr>
<td>MC IV (400)</td>
<td>53.6±1.42</td>
<td>48.9±1.64</td>
<td>118.4±3.14</td>
</tr>
<tr>
<td><strong>GA</strong> Extract treated (mg/Kg b.w)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GA II (200)</td>
<td>56.2±2.34</td>
<td>50.6±1.21</td>
<td>114.6±2.71</td>
</tr>
<tr>
<td>GA III (300)</td>
<td>55.3±4.10</td>
<td>49.1±2.51</td>
<td>117.8±2.47</td>
</tr>
<tr>
<td>GA IV (400)</td>
<td>52.7±2.71</td>
<td>50.8±2.34</td>
<td>115.6±2.74</td>
</tr>
<tr>
<td><strong>SI</strong> Extract treated (mg/Kg b.w)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SI II (200)</td>
<td>54.3±2.91</td>
<td>51.6±1.71</td>
<td>117.4±1.61</td>
</tr>
<tr>
<td>SI III (300)</td>
<td>53.7±3.01</td>
<td>48.3±1.06</td>
<td>114.6±1.01</td>
</tr>
<tr>
<td>SI IV (400)</td>
<td>54.6±2.76</td>
<td>49.7±2.11</td>
<td>116.6±2.14</td>
</tr>
<tr>
<td><strong>PP</strong> Extract treated (mg/Kg b.w)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP II (200)</td>
<td>54.8±3.12</td>
<td>47.2±2.61</td>
<td>116.7±3.01</td>
</tr>
<tr>
<td>PP III (300)</td>
<td>53.7±2.71</td>
<td>47.3±2.12</td>
<td>114.6±2.36</td>
</tr>
<tr>
<td>PP IV (400)</td>
<td>54.2±1.64</td>
<td>49.1±3.13</td>
<td>115.7±2.21</td>
</tr>
<tr>
<td>**PHF formulation treated mg/Kgb.w)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHF II (250)</td>
<td>56.3±3.10</td>
<td>50.7±1.61</td>
<td>116.2±2.11</td>
</tr>
<tr>
<td>PHF III (500)</td>
<td>57.8±2.64</td>
<td>49.1±1.75</td>
<td>115.7±3.08</td>
</tr>
</tbody>
</table>

[Transaminases - SGPT & SGOT) and Alkaline phosphatase (ALP)]
Toxicity Evaluation

Section of rat liver (X380)
(a) Normal untreated  (b) MC (400mg/Kg bw) treated, (c) GA (400mg/Kg bw) treated,
(d) SL (400mg/Kg bw) treated (e) PP (400mg/Kg bw) treated (f) PHF (500mg/Kg bw) treated
Results

6: 2: 1 ACUTE TOXICITY STUDY

In the acute toxicity studies, the aqueous extract of Melia composita Willd., Gmelina asiatica Linn., Sphaeranthus indicus Linn., Parmelia perlata Ach. and polyherbal formulation prepared out it has been subjected to toxicity studies. A single dose of 1, 2, 3, 4, 5 g/ Kg body weight (bw) were given to five different groups of animals and observed continuously for 48 hours. The plant extracts and polyherbal formulation did not showed any toxicity and mortality upto a dose of 5g/Kg bw in experimental animals. No behavioural changes were observed even in maximum dose. There were no significant variations in the initial and final body weight of the animals. The consistency of the stools and body temperatures were also found to be normal.

6: 2: 2 SHORT TERM TOXICITY STUDY

In short term toxicity study, 10% of the dosage of acute toxicity study has been taken as maximum dose and administered to experimental rats. Toxicity has been tested for three different doses - 200, 300 and 400mg/ Kg bw for selected individual plants and two different doses – 250 and 500mg/ Kg bw for formulation. Administration of plant drug and its formulation, daily for 15 days did not caused any change in general behaviour of the animals under study. Food and water intake were also normal. Body temperature, state of stools and body weight were not altered significantly by the drug treatment. The liver marker enzymes SGPT, SGOT and ALP were also not significantly altered compared to normal control (Table - 4). Histopathological studies of liver tissue showed normal hepatocytes (Plate-I). The histoarchitecture of the drug treated group of animals were also normal. The serum markers and the histology of liver tissue show the normal functioning of liver and other physiological processes. Hence 200, 300 and 400 mg/ kg bw of plant drug and 250 and 500mg/ Kg of formulation has been chosen as the dosage for studying its antidiabetic potential.
6.3 Effect of aqueous extract of *M.composita* Willd. (MC) on control and treated rats.

Effect of aqueous extract of *M.composita* Willd. (MC) on body weight of control and treated rats.

The changes in the initial and final body weight were listed in Table - 5. Observed data indicates significant improvement in the body weight (P<0.05 at 4(0mg/kgbw.) in extract treated diabetic rats with respect to diabetic control group. Group VI which received standard drug does not show any marked variations in body weight.
Figure - 13  Effect of aqueous extract of *M.composita* Willd. (MC) on body weight of control and treated rats.
Table -5: **Effect of aqueous extract of *M.composita* Willd. (MC) on body weight of control and treated rats.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Initial body weight (g)</th>
<th>Final body weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>160.6 ± 5.20</td>
<td>175.7 ± 6.25</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>161.7 ± 4.23</td>
<td>142.5 ± 3.59 <em>a</em></td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg)</td>
<td>166.2 ± 7.08</td>
<td>171.5 ± 6.14 <strong>b</strong></td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg)</td>
<td>154.7 ± 5.43</td>
<td>164.5 ± 5.05 <strong>b</strong></td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg)</td>
<td>152.2 ± 6.56</td>
<td>171.9 ± 6.71 <strong>b</strong></td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600μg/Kg)</td>
<td>153.2 ± 8.15</td>
<td>150.4 ± 7.32 <strong>c</strong></td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)  
*P < 0.05 significant when compared to normal control  
**P < 0.05 significant when compared to disease control  
*P < 0.05 significant when compared to Group II  
Statistical comparison:  
a: Group I and Group II  
**b**: Group II and Group III, IV, V  
c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*  
109
Effect of *Melia composita* Willd. (MC) on blood glucose level

The level of blood glucose in control and experimental groups after oral administration of drug for 60 days were shown in Table – 6. Diabetic animals showed progressive increase in blood sugar. Oral administration of MC leaf extract, significantly (P < 0.05) reduced the blood glucose levels. At the end of the experiment, group V animals which received 400mg/ Kg bw of plant extract showed marked decrease in the blood glucose level (98.7 ± 0.91mg/dl) compared to disease control (270.4 ± 1.24mg/dl). Reference drug – glibenclamide also showed profound decrease in blood glucose level.

Effect of *Melia composita* Willd. (MC) on serum insulin level

Figure – 14 (II) and Table – 6 shows the effect of plant extract on fasting serum insulin level in diabetic untreated and diabetic treated rats. Alloxan monohydrate as a potent chemical diabetogen causes damage to β – cells of pancreas. Hence the level of serum insulin was found to be reduced in diabetes induced group II animals (4.3 ± 0.73μU/ml) compared to normoglycemic rats (6.4 ± 1.61 μU/ml). Treatment with plant extract showed marked elevation in insulin level. Group IV and V animals which received 300 (5.6 ± 2.02 μU/ml) and 400 (6.1 ± 1.00 μU/ml) mg/kg bw respectively caused significant (P < 0.05) increase in serum insulin level.
Results

Effect of *Melia composita* Willd. (MC) on glycosylated hemoglobin (HbA1C) level

Glycated hemoglobin is used as a marker for the estimation of degree of protein glycation in diabetes mellitus. The profile of HbA1C gives the 3 months predisposition of diabetic patients. Alloxan induced hyperglycemia leads to elevated levels of HbA1C as given in Table – 6 compared to normal control. On treatment with MC extract, the level of HbA1C was seen to be reduced significantly (P < 0.05, 2.7 ± 2.26 %) compared to diabetic control (6.6 ± 2.41%). Among the three doses of plant extract – 200, 300 and 400 mg/kg bw, the maximum dose (400mg/Kg bw) showed 90 % reduction in HbA1C level. Glibenclamide causes 50 % reduction (3.1 ± 1.31%) of the HbA1C level as compared to disease control.

Effect of *Melia composita* Willd. (MC) on liver glycogen level

The effect of MC aqueous leaf extract on liver glycogen was represented in Figure - 14. Diabetic group of animals (Group II) showed significant (P < 0.05) decrease in liver glycogen level compared to normal animals. On treatment with plant extract, there was a significant increase in glycogen levels. Animals received 200 and 300mg/ Kg bw showed a moderate increase in glycogen level whereas group of rats received 400mg/Kg bw of plant extract showed a remarkable increase in liver glycogen levels.
Table 6: Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of M.composita Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Blood Sugar (mg/dl)</th>
<th>Serum insulin (μU/ml)</th>
<th>HbA1C (%)</th>
<th>Glycogen (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>86.4 ± 2.20</td>
<td>6.4 ± 1.61</td>
<td>2.6 ± 3.21</td>
<td>46.5 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>270.4 ± 1.24a*</td>
<td>4.3 ± 0.73a*</td>
<td>6.6 ± 2.41a*</td>
<td>11.7 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg)</td>
<td>241.1 ± 1.73b**</td>
<td>4.9 ± 1.89b**</td>
<td>5.3 ± 2.32b**</td>
<td>19.9 ± 1.12 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg)</td>
<td>197.1 ± 1.15b**</td>
<td>5.6 ± 2.02 b**</td>
<td>4.1 ± 1.42 b **</td>
<td>28.3 ± 2.52 b **</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg)</td>
<td>98.7 ± 0.91 b **</td>
<td>6.1 ± 1.00 b **</td>
<td>2.7 ± 2.26 b **</td>
<td>42.6 ± 0.94b **</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600μg/Kg)</td>
<td>83.4 ± 1.03c#</td>
<td>5.7 ± 1.88c#</td>
<td>3.1 ± 1.31c#</td>
<td>41.3 ± 2.86 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation

112
Results

Figure - 14 Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

I Level of blood glucose in experimental animals
II Level of serum insulin in experimental animals
III Level of HbA1C in experimental animals
IV Level of Liver glycogen in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 113
Results

Effect of *Melia composita* Willd. (MC) on the activity of glycolytic and gluconeogenetic enzymes in untreated and treated rats

Effect of aqueous extract of MC leaves on the activities of Glucokinase and Fructose – 1- 6 – bisphosphatase were depicted in Table - 7 and Figure - 15. Disease control animals showed significant alteration in the above key enzymes. The activity of Glucokinase was increased by 5, 20, and 30 % on administration of plant extract at a dosage of 200, 300 and 400mg/kg bw respectively. Fructose – 1- 6 – bisphosphatase was found to be elevated (124.7 ± 1.23 μ moles of PO₄ liberated/ min/ mg pro) significantly when compared to normal group of animals (26.4 ± 2.61 μ moles of PO₄ liberated/ min/ mg pro). Treatment of animals with MC causes remarkable decrease in fructose – 1- 6 – bisphosphatase activity. The higher dose of plant extract (400mg/ Kg bw) causes phenomenal decrease (P < 0.05) in enzyme level and in turn enzymes activity.

The activity of Glucose –6 – phosphatase and Glucose –6 – phosphate dehydrogenase on untreated and plant extract treated animals were presented graphically in Figure - 15 III & IV. Alloxan induced diabetic rats showed marked elevation (15.4 ± 1.24nmoles of Pi liberated/ min/ mg ore) in Glucose –6 – phosphatase activity, when compared to normal rats (7.7± 0.21 24nmoles of Pi liberated/ min/ mg pro). Treatment with plant extract at a dose level of 200, 300 and 400mg/ Kg bw and glibenclamide for 60 days showed significant (P < 0.05) reduction in the enzyme activity. The effect was found to be dose dependent.

MC extract showed marked variation in the activity of Glucose –6 – phosphate dehydrogenase. Diabetic rats (Group II) showed a profound decrease in enzyme activity and found to resume back to normal on administration of plant extract for 60 days. Group V animals were found to have significant (P < 0.05) increase in Glucose –6 – phosphate dehydrogenase and its effect were in par with standard drug treated (Group VI) animals.
Table – 7: Effect of aqueous extract of *M.* *composita* Willd. (MC) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Glucokinase (µ moles of Glu-6-PO₄ formed/ min/ mg protein)</th>
<th>Fructose – 1- 6-bisphosphatase (µ moles of PO₄ liberated/ min/ mg protein)</th>
<th>Glucose –6-phosphatase (nmoles of Pi liberated/ min/ mg protein)</th>
<th>Glucose – 6-phosphate dehydrogenase, (µ moles of NADPH formed/ min/ mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>136.7 ± 1.26</td>
<td>26.4 ± 2.61</td>
<td>7.7± 0.21</td>
<td>11.4 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>96.2 ± 2.05a</td>
<td>124.7 ± 1.23a</td>
<td>15.4 ± 1.24a</td>
<td>8.9 ± 0.21 a</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg)</td>
<td>100.7 ± 2.03 b</td>
<td>100.5 ± 1.90 b</td>
<td>13.6 ± 3.12b</td>
<td>9.6 ± 2.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg)</td>
<td>116.7 ± 1.78 b**</td>
<td>62.3 ± 1.52 b**</td>
<td>11.7 ± 1.85 b**</td>
<td>10.1 ± 1.56 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg)</td>
<td>129.5 ± 1.25 b**</td>
<td>31.8 ± 2.13 b**</td>
<td>9.8 ± 2.26 b**</td>
<td>10.9 ± 1.13 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>134.6 ± 2.53 c</td>
<td>32.4 ± 1.62 c</td>
<td>7.1 ± 1.37 c</td>
<td>10.0 ± 1.81 c</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure 15  Effect of aqueous extract of *M. composita* Willd. (MC) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

I  Activity of Glucokinase in experimental animals
II  Activity of Fructose-1-6-Bisphosphatase in experimental animals
III Activity of Glucose-1-6-phosphatase in experimental animals
IV Activity of Glucose-1-6-phosphate dehydrogenase in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 116
**Results**

**Effect of Melia composita Willd. (MC) on serum Lipid and lipoprotein levels**

Serum lipid profile and lipoprotein profile in treated and a diabetic rat have been given in Table – 8 & 9 and Figure – 16 & 17. Altered lipid profile occurs in diabetes mellitus and that was clearly evidenced from diabetic rats (Group II). Serum cholesterol, triglycerides, phospholipids and free fatty acids were found to be increased significantly (P < 0.05) when compared to normal rats. On treating the animals with MC aqueous extract (200, 300 and 400mg/ Kg bw) daily for 60 days caused dose dependent reduction in lipid levels. Group V animals which received 400mg/Kg bw of plant extract, showed significant (P < 0.05) elevation in cholesterol, triglycerides, phospholipids and free fatty acids when compared to diabetic control. Glibenclamide also showed significant reduction in lipid profile.

Lipoproteins LDL and VLDL were found to be elevated significantly in alloxan induced diabetic rats. Treating the animals with plant extract causes tremendous decrease (P < 0.05) in LDL and VLDL levels. The levels of HDL were also altered in diseased animals. It was found to be decreased significantly in diabetic animals (22.5 ± 2.04mg/dl) compared to normal rats (61.5± 1.42mg/ dl). MC extract was found to cause a profound influence in HDL level. It was evident from the Figure - 17 that the higher dose 400mg/Kg bw is more potent compared to other two lower doses. Reference drug was also found to cause significant variation in lipoprotein levels.
### Table - 8: Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/dl)</th>
<th>TGL (mg/dl)</th>
<th>PL (mg/dl)</th>
<th>FFA (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>60.8 ± 2.42</td>
<td>68.4 ± 1.81</td>
<td>58.2 ± 2.14</td>
<td>44.4 ± 1.52</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>141.5 ± 3.94 a*</td>
<td>168.6 ± 2.56 a*</td>
<td>122.3 ± 3.81 a*</td>
<td>110.7 ± 2.32 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>120.6 ± 2.11 b**</td>
<td>122.4 ± 1.67 b**</td>
<td>112.7 ± 1.66 b**</td>
<td>99.8 ± 2.82 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>97.6 ± 3.02 b**</td>
<td>92.8 ± 2.46 b**</td>
<td>90.7 ± 2.84 b**</td>
<td>75.2 ± 2.01 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>70.6 ± 3.12 b**</td>
<td>64.1 ± 3.02 b**</td>
<td>61.6 ± 1.75 b**</td>
<td>48.9 ± 2.14 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>59.8 ± 2.36 c*</td>
<td>64.7 ± 2.16 c*</td>
<td>62.3 ± 2.26 c*</td>
<td>47.6 ± 1.55 c*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*a* P < 0.05 significant when compared to Group II

Statistical comparison:

- a: Group I and Group II
- b: Group II and Group III, IV, V
- c: Group II and VI.

*Results*

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*

118
Figure - 16  Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

| I | Level of cholesterol in experimental animals |
| II | Level of triglyceride in experimental animals |
| III | Level of phospholipids in experimental animals |
| IV | Level of free fatty acids in experimental animals |
### Table - 9: Serum lipoprotein profile of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL - Cholesterol (mg/dl)</th>
<th>LDL - Cholesterol (mg/dl)</th>
<th>VLDL - Cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>61.5 ± 1.42</td>
<td>41.5 ± 2.01</td>
<td>13.9 ± 3.14</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>22.5 ± 2.04 a*</td>
<td>69.3 ± 1.52 a*</td>
<td>30.3 ± 1.01 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>30.6 ± 1.72b**</td>
<td>53.4 ± 3.16 b**</td>
<td>25.2 ± 3.02 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>43.7 ± 2.28 b**</td>
<td>47.2 ± 2.62 b**</td>
<td>20.3 ± 0.64 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>53.7 ± 2.41 b**</td>
<td>42.8 ± 1.95 b**</td>
<td>15.6 ± 1.35 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>52.5 ± 1.08 cº</td>
<td>44.7 ± 2.64 cº</td>
<td>17.2 ± 2.07 cº</td>
</tr>
</tbody>
</table>

[**HDL - cholesterol, LDL - cholesterol and VLDL - cholesterol**]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

ºP < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation
Figure - 17 Serum lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

I Level of HDL-cholesterol in experimental animals

II Level of LDL-cholesterol in experimental animals

III Level of VLDL-cholesterol in experimental animals
**Results**

**Effect of Melia composita Willd. (MC) on tissue lipid and lipoprotein levels**

Effect of plant extract on tissue lipid profile was tabulated in 10 and presented graphically in Figure - 18. Tissue lipids are the source of energy in severe diabetes mellitus and hence increased levels of tissue cholesterol, triglycerides, phospholipids and free fatty acids were seen in diabetic control (Group II). Whereas treating the animals with plant extract causes significant (P < 0.05) reduction in lipid profile. This is due to the effect of plant extract which causes increased insulin secretion and thereby increased uptake of glucose by the tissues. The antihyperlipidemic effect was found to be dose dependent. Animals received 400mg/Kg bw of plant drug showed remarkable decrease in cholesterol, triglycerides, phospholipids and free fatty acid levels, which was nearer to normal values.

Tissue lipoprotein level was also altered significantly in diabetic animals. There was a profound increase in LDL and VLDL levels and a notable decrease in HDL levels. The altered levels of lipoproteins were restored to normal on treating the diabetic animals with aqueous extract at dose levels of 200, 300 and 400mg/Kg bw. Among the three doses, animals which received the higher dose (400mg/Kg bw) showed significant reduction in LDL and VLDL levels and increase in HDL levels.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Table 10: Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/100g wet tissue)</th>
<th>TGL (mg/100g wet tissue)</th>
<th>PL (mg/100g wet tissue)</th>
<th>FFA (mg/100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>78.1 ± 1.42</td>
<td>65.2 ± 2.01</td>
<td>72.8 ± 1.14</td>
<td>58.2 ± 1.32</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>172.5 ± 2.05 a*</td>
<td>152.1 ± 2.32 a*</td>
<td>132.8 ± 2.81 a*</td>
<td>111.2 ± 3.42 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>142.5 ± 3.12 b**</td>
<td>117.6 ± 1.65 b**</td>
<td>121.6 ± 2.80 b**</td>
<td>95.3 ± 3.02 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>102.8 ± 1.22 b**</td>
<td>96.5 ± 1.52 b**</td>
<td>102.0 ± 1.52 b**</td>
<td>84.2 ± 1.57 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>81.5 ± 2.21 b**</td>
<td>69.2 ± 1.39 b**</td>
<td>77.6 ± 3.15 b**</td>
<td>61.4 ± 2.09 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600mg/Kg bw)</td>
<td>89.8 ± 2.72 c#</td>
<td>71.8 ± 2.04 c#</td>
<td>79.7 ± 2.52 c#</td>
<td>65.1 ± 1.54 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure - 18  Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *M.composita* Willd. (MC) for 60 days.

I

II

III

IV

I  Level of cholesterol in experimental animals
II  Level of triglyceride in experimental animals
III  Level of phospholipids in experimental animals
IV  Level of free fatty acids in experimental animals
Table – 11: Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/ 100g wet tissue)</th>
<th>LDL -Cholesterol (mg/ 100g wet tissue)</th>
<th>VLDL -Cholesterol (mg/ 100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>45.2 ± 2.42</td>
<td>50.6 ± 2.61</td>
<td>15.9 ± 3.24</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>20.2 ± 2.84 a*</td>
<td>79.3 ± 1.22 a*</td>
<td>35.5 ± 1.56 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>26.4 ± 2.51b**</td>
<td>71.2 ± 2.35 b**</td>
<td>30.5 ± 2.02 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>32.3 ± 3.13 b**</td>
<td>64.2 ± 2.05 b**</td>
<td>25.2 ± 2.12 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>42.9 ± 2.32 b**</td>
<td>54.3 ± 3.20 b**</td>
<td>17.2 ± 2.32 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>50.2 ± 1.23 c#</td>
<td>54.7 ± 3.24 c#</td>
<td>18.0 ± 0.79 c#</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Figure -19  Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *M.composita* Willd. (MC) for 60 days.

I

II

III

I  Level of HDL- cholesterol in experimental animals

II  Level of LDL- cholesterol in experimental animals

III  Level of VLDL- cholesterol in experimental animals
Effect of *Melia composita* Willd. (MC) on serum protein, transaminases (SGPT and SGOT), alkaline phosphatases and bilirubin

Table 12 shows the effect of MC extract in serum liver marker enzymes – SGPT, SGOT, ALP and protein. In diabetes mellitus, due to impaired carbohydrate metabolism, alternative source of energy is utilized by the cells. Hence there was a profound decrease in serum protein level in diabetic group (Group II) of animals. On treating the diabetic animals with MC extract, the protein is spared for tissue building and hence significant (P < 0.05) increase in protein was seen.

Alloxan causes profound alteration in transaminases ALP and bilirubin. Treatment with plant extract causes significant (P < 0.05) reduction in these serum liver marker enzymes and elevation in bilirubin levels. The enzyme activities were brought back to near normal and the effect was found to be dose dependent. Hence group V animals which received 400mg/Kg bw showed profound enzyme activity compared to group III and IV animals. Glibenclamide also had influence in enzyme activity.
**Table – 12: Effect of aqueous extract of *M.composita* Willd. (MC) on Serum liver markers in untreated and treated rats.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/dl)</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>58.9 ±3.02</td>
<td>63.7 ±1.23</td>
<td>48.9 ±1.54</td>
<td>120.6 ±3.23</td>
<td>0.40 ±2.51</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>35.1 ±1.04a*</td>
<td>129.6 ±2.82 a*</td>
<td>123.7±1.39a*</td>
<td>217.8 ±2.41 a*</td>
<td>1.96 ±1.21a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>40.7 ±1.35 **</td>
<td>119.5 ±3.10 h**</td>
<td>102.3±2.52 h**</td>
<td>192.3 ±2.42 a**</td>
<td>1.49 ±1.81 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>44.7 ±2.08 b**</td>
<td>101.5±2.23 b**</td>
<td>80.6±1.94 b**</td>
<td>164.6±1.02 a**</td>
<td>1.02 ±3.02 a**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>53.9±2.12 b**</td>
<td>80.1±1.85 b**</td>
<td>51.5±1.05 b**</td>
<td>137.7±2.56 a**</td>
<td>0.64±1.36 a**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>55.7±2.65 c#</td>
<td>71.7±1.03 c#</td>
<td>52.6±1.27 c#</td>
<td>136.7±2.52 c#</td>
<td>0.61±1.20 c#</td>
</tr>
</tbody>
</table>

[Protein, transaminases (SGPT and SGOT), alkaline phosphatase (ALP), and bilirubin]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Figure - 20  Effect of aqueous extract of *M.composita* Willd. (MC) on Serum liver markers in untreated and treated rats.

I  Level of protein in experimental animals
II  Activity of SGPT in experimental animals
III Activity of SGOT in experimental animals
IV  Activity of ALP in experimental animals
V  Level of bilirubin in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
**Results**

**Effect of Melia composita Willd. (MC) on antioxidant status of experimental animals**

Table 13 & Fig.21 depicts the antioxidant status of the experimental animals. Alloxan causes damage to beta cell of langerhans by producing reactive oxygen species. This oxidative damage causes significant (P < 0.05) increase in lipid peroxidation; decrease in reduced glutathione level; and decrease in activity of superoxide dismutase and catalase. On treatment with plant extract helps the diabetic animals to resume back to normal Group V animals which received 400mg/Kg bw showed increased level of GSH and decreased level of peroxides. Activity of SOD and catalase activity were also enhanced on plant drug treated animals. Glibenclamide was found to cause only mild influence in enzymatic and non enzymatic antioxidants.
Table 13: Effect of aqueous extract of M. composita Willd. (MC) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Protein (mg/100g wet tissue)</th>
<th>LPO (μM of MDA/mg protein)</th>
<th>Catalase (nM of hydrogen peroxide decomposed/min/mg protein)</th>
<th>SOD (U/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>I</td>
<td>II</td>
<td>III</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Normal control</td>
<td>Diabetic control</td>
<td>MC treated (200mg/Kg bw)</td>
<td>Diabetic control</td>
</tr>
<tr>
<td></td>
<td>42.5±2.22</td>
<td>10.4±2.43</td>
<td>31.5±2.68*</td>
<td>34.3±1.94**</td>
</tr>
<tr>
<td></td>
<td>4.9±1.1</td>
<td>9.9±1.21</td>
<td>3.1±1.2 a**</td>
<td>13.1±1.02 a**</td>
</tr>
<tr>
<td></td>
<td>21.6±1.23</td>
<td>21.6±1.23</td>
<td>21.6±1.23</td>
<td>21.6±1.23</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control
**P < 0.01 significant when compared to disease control

Statistical comparison:
a. Group I and Group II
b. Group I and Group III, IV, V

c. Group II and VI.

[Superoxide dismutase (SOD), Catalase, lipid peroxidation (LPO), and glutathione (GSH)]
**Results**

Figure - 21 Effect of aqueous extract of *M. composita* Willd. (MC) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

I

II

III

IV

V

I Level of protein in experimental animals

II Level of LPO in experimental animals

III Level of GSH in experimental animals

IV Activity of SOD in experimental animals

V Activity of catalase in experimental animals

_A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation_ 132
Effect of *Melia composita* Willd. (MC) on serum urea, uric acid and creatinine

Effect of MC leaf extract on serum urea, uric acid and creatinine were presented in Table 14 & Fig.22. The levels of urea, uric acid and creatinine were found to be increased in diabetic animals (95.6 ± 2.14, 0.78 ± 2.42, and 4.0± 1.25 mg/ dl respectively) compared to normal group of rats (38.6± 1.62, 0.43 ± 1.21, 0.12 ± 2.04 mg/ dl). Treatment of diabetic animals with plant extract for 60 days causes significant reduction (P < 0.05) in the urea, uric acid and creatinine levels. Group V animals showed profound decrease in all the above three parameters. Group IV animals which received standard drug show mild influence in urea, uric acid and creatinine levels.
Table – 14: Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Urea (mg/dl)</th>
<th>Uric acid (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>38.6 ± 1.62</td>
<td>0.43 ± 1.21</td>
<td>0.12 ± 2.04</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>95.6 ± 2.14 a*</td>
<td>0.78 ± 2.42 a*</td>
<td>4.0 ± 1.25 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + MC treated (200mg/Kg bw)</td>
<td>82.45 ± 2.52b**</td>
<td>0.68 ± 1.16 b**</td>
<td>3.1 ± 1.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + MC treated (300mg/Kg bw)</td>
<td>62.3 ± 1.29 b**</td>
<td>0.54 ± 1.23 b**</td>
<td>2.3 ± 2.45 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + MC treated (400mg/Kg bw)</td>
<td>43.2 ± 1.32b**</td>
<td>0.45 ± 1.55 b**</td>
<td>1.1 ± 1.51 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic +Glibenclamide treated (600µg/Kg bw)</td>
<td>40.2 ± 1.97c*</td>
<td>0.49 ± 2.04 c*</td>
<td>0.48 ± 2.23 c*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Figure - 22  Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *M. composita* Willd. (MC) for 60 days.

I Level of urea in experimental animals

II Level of uric acid in experimental animals

III Level of creatinine in experimental animals

*A Study On Antidiabetic Potential Of Selected Herbs And A Herbal Formulation*
Results

Effect of *Melia composita* Willd (MC) and their formulation on histoarchitecture of pancreas:

The haematoxylin and eosin stained sections of normal rat pancreatic tissue showed intact and round or elongated islets of langerhans (Plate-IIa) with granulated beta cells appearing darker. Small, shrunken islets and destruction of beta cells were observed in diabetic untreated animals (Plate-IIb) MC extracts treated animals showed a profound enhancement in the size and shape of islets of langerhans. Among the drug treated animals, group of animals which received maximum dose (400mg/Kg bw) showed better granulation and rejuvenation of β – cells (Plate-IIIc).
Section of rat pancreas (x380)

a - Normal rat  b - diseased diabetic rat  c - glibenclamide treated rat.

IS - Islets of langerhans, VD - Vascular degeneration, MD - moderate degeneratio
Photomicrographs of Histopathological Studies

MC treated

Section of rat pancreas (x380)

a - 200mg/Kg MC treated  b - 300mg/Kg MC treated  c - 400mg/Kg MC treated

IS - Islets of langerhans, GI - Granulated islet, MR - moderate regeneration.
6.4 Effect of aqueous extract of *Gmelina asiatica* Linn. (GA) on drug treated and untreated diabetic animals:

**Effect of *Gmelina asiatica* Linn. (GA) on body weight of experimental animals**

Variation in the initial and final body weight in drug treated and untreated animals were presented in Table – 15 and Figure – 23. In untreated diabetic control there was a significant reduction in body weight, whereas animals treated with GA aqueous extract showed significant (p<0.05) increase in body weight. GS at a dose level of 400mg/Kg bw showed statistically significant increase in final body weight compared to disease control.
Table – 15: **Effect of aqueous extract of G.asiatica L (GA) on body weight of control and treated rats.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Initial body weight (g)</th>
<th>Final body weight(g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>160.6 ± 5.20</td>
<td>175.7 ± 6.25</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>161.7 ± 4.23</td>
<td>142.5 ± 3.59 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg)</td>
<td>156.3 ± 6.18</td>
<td>164.3 ± 5.08 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg)</td>
<td>154.2 ± 5.43</td>
<td>162.1 ± 4.35 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg)</td>
<td>162.2 ± 4.56</td>
<td>178.9 ± 6.55 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenelamide (600μg/Kg)</td>
<td>153.2 ± 8.15</td>
<td>150.4 ± 7.32 c&quot;</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Figure – 23  Effect of aqueous extract of *G. asiatica* L (GA) on body weight profile of control and treated rats.
Results

Effect of *Gmelina asiatica* Linn. (GA) on blood glucose level

Effect of GA in blood glucose level was shown in Table - 16 & Figure- 24(I). Experimental induction of diabetes mellitus caused severe hyperglycemia which was evident from the mean blood glucose level of group II disease control animals. Treatment with test drug showed significant (p<0.05) decrease in the blood glucose level. GA extract at a dose level of 200, 300 and 400mg/Kg bw reduced the glucose level (194.9 ± 1.45, 108.4 ± 1.62 and 89.7 ± 1.91mg/dl respectively) compared to diabetic untreated animals. Group VI animals which received 600μg/Kg bw of glibenclamide also caused significant decrease in blood glucose level.

Effect of *Gmelina asiatica* Linn. (GA) on serum insulin level

Table – 16 & Figure- 24(II) shows the effect of GA on serum insulin levels in treated and untreated rats. Group II alloxonized animals showed profound decrease (4.3 ± 0.73 μU/ml) in the insulin level compared to normal control (6.4 ± 1.61 μU/ml). Treatment with plant extract were found to restore the normal insulin secretion which was evident from the significant (p<0.05) increase in the serum insulin levels found in Group III (5.0 ± 0.44 μU/ml), IV (5.9 ± 0.92 μU/ml) and V (6.6 ± 1.90 μU/ml) animals. Glibenclamide treated animals also showed increased level of insulin.
Results

Effect of Gmelina asiatica Linn. (GA) on glycosylated hemoglobin (HbA1C) level

Levels of HbA1C in normal, diabetic and drug treated animals were given in Table – 16 & Figure- 24(II). The blood glucose level was elevated on alloxan induction which results in glycation of hemoglobin. Hence there was significant increase in the HbA1C levels in diabetic rats but on treatment with GA extract the levels were brought back to near normal. The effect of plant extract was found to be dose dependent. Reference drug also decreases the HbA1C levels significantly (p<0.05) compared to diabetic control.

Effect of Gmelina asiatica Linn. (GA) on liver glycogen level

Table – 16 & Figure- 24(IV) shows the effect of plant extract on liver glycogen levels. Administration of plant extract for 60 days increases the glycogen level significantly. Animals (Group V) which received maximum dose (400mg/Kg bw) were found to have profound increase (43.5 ± 1.82mg/g tissue) in the glycogen level. Whereas disease control animals which were untreated (Group II- 11.7 ± 0.21 mg/g issue) had very low level of liver glycogen compared to normal animals (Group I - 46.5 ± 0.91 mg/g tissue).
Table- 16: Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *G.asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Blood Sugar (mg/dl)</th>
<th>Serum insulin (μU/ml)</th>
<th>HbA1C (%)</th>
<th>Glycogen (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>86.4 ± 2.20</td>
<td>6.4 ± 1.61</td>
<td>2.6 ± 3.21</td>
<td>46.5 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>270.4 ± 1.24*</td>
<td>4.3 ± 0.73a*</td>
<td>6.6 ± 2.41a*</td>
<td>11.7 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg)</td>
<td>194.9 ± 1.45b**</td>
<td>5.0 ± 0.44b**</td>
<td>5.4 ± 0.82b**</td>
<td>21.5 ± 1.12 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg)</td>
<td>108.4 ± 1.62b**</td>
<td>5.9 ± 0.92 b**</td>
<td>3.6 ± 2.30 b**</td>
<td>34.6 ± 0.12 b **</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg)</td>
<td>89.7 ± 1.91 b **</td>
<td>6.6 ± 1.90 b **</td>
<td>2.1 ± 2.18 b **</td>
<td>43.5 ± 1.82 b **</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide</td>
<td>83.4 ± 1.53c#</td>
<td>5.7 ± 1.58c#</td>
<td>3.1 ± 1.21c#</td>
<td>41.3 ± 1.76 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

---

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Figure - 24  Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

I Level of blood glucose in experimental animals  
II Level of serum insulin in experimental animals  
III Level of HbA1C in experimental animals  
IV Level of Liver glycogen in experimental animals
Results

Effect of Gmelina asiatica Linn. (GA) on the activity of glycolytic and gluconeogenetic enzymes in untreated and treated rats

Effect of aqueous extract of GA leaves on the activities of Glucokinase and The activity of glucokinase were reduced in disease control animals and resumed back to normal on treatment with GA plant aqueous extract at various doses. Fructose - 1- 6 - bisphosphatase were depicted in Table - 17 and Figure - 25. Disease control animals showed significant alteration in the above enzymes. Fructose – 1- 6 – bisphosphatase was found to be elevated \( (124.7 \pm 1.23 \text{ μ moles of PO}_4 \text{ liberated/ min/ mg pro}) \) significantly when compared to normal group of animals \( (26.4 \pm 2.61 \text{ μ moles of PO}_4 \text{ liberated/ min/ mg pro}) \). Treatment of animals with GA caused remarkable decrease in fructose – 1- 6 – bisphosphatase activity. The higher dose of plant extract \( (400\text{mg/ Kg bw}) \) caused phenomenal decrease \( (P < 0.05) \) in enzyme level and in turn enzymes activity.

The activity of Glucose – 6 – phosphatase and Glucose – 6 – phosphate dehydrogenase on untreated and plant extract treated animals were presented graphically in Figure - 25III & IV. Alloxan induced diabetic rats showed marked elevation \( (15.4 \pm 1.24\text{nmoles of Pi liberated/ min/ mg pro}) \) in Glucose – 6 – phosphatase activity, when compared to normal rats \( (7.7 \pm 0.21 \text{24nmoles of Pi liberated/ min/ mg pro}) \). Treatment with plant extract at a dose level of 200, 300 and 400mg/ Kg bw and glibenclamide for 60 days showed significant \( (P < 0.05) \) reduction in the enzyme activity. The effect was found to be dose dependent.

GA extract showed marked variation in the activity of Glucose – 6 – phosphate dehydrogenase. Diabetic rats (Group II) showed a profound decrease in enzyme activity and found to resume back to normal on administration of plant extract for 60 days. Group V animals were found to have significant \( (P < 0.05) \) increase in Glucose – 6 – phosphate dehydrogenase and its effect were in par with standard drug treated (Group VI) animals.
Table – 17: Effect of aqueous extract of *G. asiatica* L (GA) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Glucokinase (μ moles of Glu-6-P formed/min/mgprotein)</th>
<th>Fructose – 1- 6 - bisphosphatase (μ moles of PO₄ liberated/ min/ mg protein)</th>
<th>Glucose –6 – phosphatase (mnoles of Pi liberated/ min/ mg protein)</th>
<th>Glucose – 6 – phosphate dehydrogenase, (μ moles of NADPH formed/ min/ mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>136.7 ± 1.26</td>
<td>26.4 ± 2.61</td>
<td>7.7 ± 0.21</td>
<td>11.4 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>96.2 ± 2.05a*</td>
<td>124.7 ± 1.23a*</td>
<td>15.4 ± 1.24a*</td>
<td>8.9 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg)</td>
<td>110.6 ± 1.43 b**</td>
<td>90.2± 1.40 b**</td>
<td>11.7 ±1.23b**</td>
<td>9.9 ± 1.12 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg)</td>
<td>121.3 ± 1.67 b**</td>
<td>52.1± 0.81 b**</td>
<td>9.6 ± 2.05 b**</td>
<td>10.4 ± 0.12 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg)</td>
<td>133.8 ± 0.91 b**</td>
<td>30.1± 1.20 b**</td>
<td>7.8 ± 1.15 b**</td>
<td>11.7 ± 1.82 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600μg/Kg)</td>
<td>134.6 ± 2.05 c#</td>
<td>32.4± 1.62 c#</td>
<td>7.1 ± 0.27 c#</td>
<td>10.0 ± 1.76 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 145
Results

Figure - 25 Effect of aqueous extract of *G. asiatica* L (GA) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

I

II

III

IV

| I | Activity of Glucokinase in experimental animals |
| II | Activity of Fructose-1-6-Bisphosphatase in experimental animals |
| III | Activity of Glucose-1-6-phosphatase in experimental animals |
| IV | Activity of Glucose-1-6-phosphate dehydrogenase in experimental animals |

_A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation_ 146
Results

Effect of *Gmelina asiatica* Linn. (GA) on serum Lipid and lipoprotein levels

Table 18 and 19 & Figure 26 and 27 depicts the serum lipid and lipoproteins levels. Diabetic animals showed elevated levels of cholesterol (141.5 ± 3.94 mg/Kg bw), TGL (168.6 ± 2.56 mg/Kg bw), PL (122.3 ± 3.81 mg/Kg bw) and FFA (110.7± 2.32 mg/Kg bw) compared to normal animals. Aqueous extract of GA were found to have a profound influence on the serum lipid profiles. Animals which received 400mg/Kg bw showed remarkable decrease in the serum lipid profile compared to other dose level (200 and 300mg/Kg bw) of GA extract.

Lipoprotein levels were also found to be altered in disease control significantly (P < 0.05) compared to normal animals. The levels of LDL and VLDL which were elevated in disease control were found to be decreased in plant extract treated animals. Group V animals that received 400mg/ Kg bw showed profound decrease in the levels of LDL (40.6 ± 1.95mg/dl) and VLDL (14.2 ± 2.85 mg/dl) compared to disease control (LDL - 69.3 ± 1.52mg/dl and VLDL - 30.3 ± 1.01mg/dl). HDL level in disease control animals (22.5 ± 2.04mg/dl) were decreased significantly compared to normal rats (61.5 ± 1.42mg/dl). Treatment with test drug elevated the serum HDL levels significantly and the effects were found to be dose dependent.
Table – 18: Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/dl)</th>
<th>TGL (mg/dl)</th>
<th>PL (mg/dl)</th>
<th>FFA (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>60.8 ± 2.42</td>
<td>68.4 ± 1.81</td>
<td>58.2 ± 2.14</td>
<td>44.4 ± 1.52</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>141.5 ± 3.94 a*</td>
<td>168.6 ± 2.56 a*</td>
<td>122.3 ± 3.81 a*</td>
<td>110.7 ± 2.32 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>117.4 ± 1.61 b**</td>
<td>114.4 ± 2.91 b**</td>
<td>108.7 ± 1.76 b**</td>
<td>98.5 ± 3.80 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>93.7 ± 1.35 b**</td>
<td>84.6 ± 1.25 b**</td>
<td>81.6 ± 2.54 b**</td>
<td>65.7 ± 1.91 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>63.4 ± 3.74 b**</td>
<td>63.1 ± 1.27 b**</td>
<td>60.4 ± 2.05 b**</td>
<td>42.3 ± 1.04 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>59.8 ± 4.10 c #</td>
<td>64.7 ± 2.34 c #</td>
<td>62.3 ± 1.47 c #</td>
<td>47.6 ± 1.65 c #</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 26 Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

I

- Group I
- Group II
- Group III
- Group IV
- Group V
- Group VI

II

- Group I
- Group II
- Group III
- Group IV
- Group V
- Group VI

III

- Group I
- Group II
- Group III
- Group IV
- Group V
- Group VI

IV

- Group I
- Group II
- Group III
- Group IV
- Group V
- Group VI

I Level of cholesterol in experimental animals
II Level of triglyceride in experimental animals
III Level of phospholipids in experimental animals
IV Level of free fatty acids in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 149
Table – 19: Serum lipoprotein profile of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/dl)</th>
<th>LDL -Cholesterol (mg/dl)</th>
<th>VLDL -Cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>61.5 ± 1.42</td>
<td>41.5 ± 2.01</td>
<td>13.9 ± 3.14</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>22.5 ± 2.04 a*</td>
<td>69.3 ± 1.52 a*</td>
<td>30.3 ± 1.01 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>31.4 ± 2.62b**</td>
<td>52.7 ± 2.56 b**</td>
<td>24.6 ± 2.12 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>49.5 ± 1.02 b**</td>
<td>45.5 ± 1.05 b**</td>
<td>17.3 ± 1.64 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>56.7 ± 2.32 b**</td>
<td>40.6 ± 1.95 b**</td>
<td>14.2 ± 2.85 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>52.5 ± 1.28 c #</td>
<td>44.7 ± 3.44 c #</td>
<td>17.2 ± 1.27 c #</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)  
*P < 0.05 significant when compared to normal control  
**P < 0.05 significant when compared to disease control  
#P < 0.05 significant when compared to Group II  
Statistical comparison:  
a: Group I and Group II  
b: Group II and Group III, IV, V  
c: Group II and VI.
Results

Figure - 27  Serum lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *G.asiatica* L. (GA) for 60 days.

I

II

III

I  Level of HDL- cholesterol in experimental animals
II  Level of LDL- cholesterol in experimental animals
III  Level of VLDL- cholesterol in experimental animals
Results

Effect of *Gmelina asiatica* Linn. (GA) on tissue lipid and lipoprotein levels

Effect of plant extract on tissue lipid profile was tabulated in 20 & 21 and presented graphically in Figure – 28 & 29. In severe diabetes mellitus, increased levels of tissue cholesterol, triglycerides, phospholipids and free fatty acids were seen. Whereas treating the animals with plant extract causes significant (P < 0.05) reduction in lipid profile. The effect was found to be dose dependent. Animals received 400mg/Kg bw of plant drug showed remarkable decrease in cholesterol, triglycerides, phospholipids and free fatty acid levels, which was nearer to normal values.

Tissue lipoprotein level was also altered significantly in diabetic animals. There was a profound increase in LDL and VLDL levels and a notable decrease in HDL levels. The altered levels of lipoproteins were resumed to normal on treating the diabetic animals with aqueous extract at dose levels of 200, 300 and 400mg/Kg bw.
Table - 20: Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/ 100g wet tissue)</th>
<th>TGL (mg/100g wet tissue)</th>
<th>PL (mg/ 100g wet tissue)</th>
<th>FFA (mg/ 100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>78.1 ± 1.42</td>
<td>65.2 ± 2.01</td>
<td>72.8 ± 1.14</td>
<td>58.2 ± 1.32</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>172.5 ± 2.05 a*</td>
<td>152.1 ± 2.32 a*</td>
<td>132.8 ± 2.81 a*</td>
<td>111.2 ± 3.42 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>137.4 ± 2.55b**</td>
<td>112.4 ± 2.36 b**</td>
<td>110.1 ± 3.26 b**</td>
<td>91.3 ± 3.52 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>97.2 ± 1.08 b**</td>
<td>85.2 ± 1.02 b**</td>
<td>92.0 ± 1.04 b**</td>
<td>64.7 ± 1.01 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>71.4± 2.14 b**</td>
<td>66.5 ± 1.65 b**</td>
<td>76.5± 1.01 b**</td>
<td>59.2 ± 1.59 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>89.8 ± 2.86 c&quot;</td>
<td>71.8 ± 2.94 c&quot;</td>
<td>79.7± 1.55 c&quot;</td>
<td>65.1± 2.34 c&quot;</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 153
Figure - 28 Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

I Level of cholesterol in experimental animals
II Level of triglyceride in experimental animals
III Level of phospholipids in experimental animals
IV Level of free fatty acids in experimental animals
Table – 21: Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *G.asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL –Cholesterol (mg/ 100g wet tissue)</th>
<th>LDL –Cholesterol (mg/ 100g wet tissue)</th>
<th>VLDL –Cholesterol (mg/ 100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>45.2 ± 2.42</td>
<td>50.6 ± 2.61</td>
<td>15.9 ± 3.24</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>20.2 ± 2.84 a*</td>
<td>79.3 ± 1.22 a*</td>
<td>35.5 ± 1.56 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>29.3 ± 3.62b**</td>
<td>68.9 ± 2.16 b**</td>
<td>27.8 ± 1.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>39.6 ± 3.02 b**</td>
<td>59.3 ± 2.25 b**</td>
<td>19.8 ± 1.52 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>49.9 ± 1.52 b**</td>
<td>51.2 ± 3.05 b**</td>
<td>15.2 ± 2.02 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>50.2 ± 1.90 c#</td>
<td>54.7 ± 3.14 c#</td>
<td>18.0 ± 1.79 c#</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
**Results**

Figure - 29  Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *G.asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>I</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>Group IV</td>
<td>Group V</td>
<td>Group VI</td>
</tr>
</tbody>
</table>

| Level of HDL- cholesterol in experimental animals |
| Level of LDL- cholesterol in experimental animals |
| Level of VLDL- cholesterol in experimental animals |

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 156
**Effect of *Gmelina asiatica* Linn. (GA) on serum protein, transaminases (SGPT and SGOT) alkaline phosphatases, and bilirubin**

Effect of plant on serum markers were presented in **Table 22 & figure 30.** The serum protein were found to be decrease in disease control animals and on treatment with plant extract an glibenclamide, the level were found to be elevated significantly (P < 0.05). The activity of serum markers were altered significantly on alloxan induction (Group II). Extract of test drug decreased the levels of GPT, GOT, ALP and Bilirubin significantly. The effects were found to be dose dependent. Glibenclamide also showed moderate influence on the serum markers but not as effective as plant extract treated animals.
Table – 22: Effect of aqueous extract of *G. aslatica* L. (GA) on Serum liver markers in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/dl)</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>58.9±3.02</td>
<td>63.7±1.23</td>
<td>48.9±1.54</td>
<td>120.6±3.23</td>
<td>0.40±2.51</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>35.1±1.04a*</td>
<td>129.6±2.82a*</td>
<td>123.7±1.39a*</td>
<td>217.8±2.41a*</td>
<td>1.96±1.21a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>42.7±1.24 **</td>
<td>106.9±3.06 b**</td>
<td>98.5±3.72 b**</td>
<td>182.5±2.09 a**</td>
<td>1.09±1.01 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>47.4±1.58 b**</td>
<td>80.7±2.55 b**</td>
<td>72.3±2.04 b**</td>
<td>152.7±1.56 a**</td>
<td>0.73±3.45 a*</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>57.1±3.02 b**</td>
<td>64.3±2.35 b**</td>
<td>50.1±1.85 b**</td>
<td>131.7±2.09 a**</td>
<td>0.41±1.01 a**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>55.7±2.57 c#</td>
<td>71.7±1.28c#</td>
<td>52.6±0.97 c#</td>
<td>136.7±4.67 c#</td>
<td>0.61±1.20 c#</td>
</tr>
</tbody>
</table>

[protein, transaminases (SGPT and SGOT), alkaline phosphatase (ALP), and bilirubin]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 30  Effect of aqueous extract of *G. asiatica* L. (GA) on Serum liver markers in untreated and treated rats.

I  
II  
III  
IV  
V  

I  Level of protein in experimental animals  
II  Activity of SGPT in experimental animals  
III  Activity of SGOT in experimental animals  
IV  Activity of ALP in experimental animals  
V  Level of bilirubin in experimental animals  

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 159
Results

Effect of *Gmelina asiatica* Linn. (GA) on antioxidant status of experimental animals

Induction of alloxan at a dose level of 150mg/Kg bw causes oxidative damage to the pancreatic beta cells. This leads to profound alterations in the antioxidant status of the diseased animals. Treatment with the plant extract at dose levels of 200, 300 and 400mg/Kg bw restored 90% of the normal antioxidant activities. The lipidperoxide levels were reduced, GSH levels were increased, activities of SOD and catalase were increased significantly (Table 23 & figure 31). The effects of test drug on enzymatic and non enzymatic antioxidants were found to be dose dependent. Group VI animals which received standard drug, glibenclamide also maintains antioxidant status but not in par with plant extract treated animals.
Table – 23: Effect of aqueous extract of *G. asiatica* L. (GA) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/100g wet tissue)</th>
<th>LPO (nM of MDA/mg protein)</th>
<th>GSH (nM/mg protein)</th>
<th>SOD (IU/mg protein)</th>
<th>Catalase (nM of hydrogen peroxide decomposed/min/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>42.5±2.22</td>
<td>10.4±2.43</td>
<td>34.7±2.03</td>
<td>21.6±1.23</td>
<td>50.4±1.21</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>31.5 ± 2.68*</td>
<td>25.6±1.02 a*</td>
<td>18.9±1.02a*</td>
<td>9.9±1.21 a*</td>
<td>24.6±3.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic+ GA treated (200mg/Kg bw)</td>
<td>34.5 ±1.04 **</td>
<td>19.6±2.23 b**</td>
<td>21.6±2.45 b**</td>
<td>11.7± 1.22 a**</td>
<td>32.1±4.01 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic+ GA treated (300mg/Kg bw)</td>
<td>39.2±2.05 b**</td>
<td>14.3±1.51 b**</td>
<td>26.2±2.15 b**</td>
<td>14.2±2.06 a**</td>
<td>39.5±2.06 a**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic+ GA treated (400mg/Kg bw)</td>
<td>43.2±3.82 b**</td>
<td>10.1±1.27 b**</td>
<td>32.6±1.03 b**</td>
<td>20.6± 2.21 a**</td>
<td>46.2±3.11 a**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>36.7±3.07 c*</td>
<td>14.7±2.08c*</td>
<td>29.7±1.12c*</td>
<td>16.6± 2.12 c*</td>
<td>42.2±2.12 c*</td>
</tr>
</tbody>
</table>

[Superoxide dismutase (SOD) Catalase, lipid peroxidation (LPO) and glutathione (GSH)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 31  Effect of aqueous extract of *G. asiatica* L. (GA) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

I  
II  
III  
IV  
V  

I  Level of protein in experimental animals  
II  Level of LPO in experimental animals  
III  Level of GSH in experimental animals  
IV  Activity of SOD in experimental animals  
V  Activity of catalase in experimental animals
Effect of *Gmelina asiatica* Linn. (GA) on serum urea, uric acid and creatinine

Effect of MC leaf extract on serum urea, uric acid and creatinine were presented in Table 24 & figure 32. The levels of urea, uric acid and creatinine were found to be increased in diabetic animals (95.6 ± 2.14, 0.78 ± 2.42, and 4.0± 1.25 mg/ dl respectively) compared to normal group of rats (38.6± 1.62, 0.43 ± 1.21, 0.12 ± 2.04 mg/ dl). Treatment of diabetic animals with plant extract for 60 days caused significant reduction (P < 0.05) in the urea, uric acid and creatinine levels. Group V animals which received 400mg/Kg bw showed profound decrease in all the above three parameters. Group IV animals which received standard drug show mild influence in urea, uric acid and creatinine levels.
Table – 24: Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *G. asiatica* L. (GA) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Urea (mg/dl)</th>
<th>Uric acid (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>38.6 ± 1.62</td>
<td>0.43 ± 1.21</td>
<td>0.12 ± 2.04</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>95.6 ± 2.14 a*</td>
<td>0.78 ± 2.42 a*</td>
<td>4.0 ± 1.25 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + GA treated (200mg/Kg bw)</td>
<td>76.4 ± 2.02b**</td>
<td>0.55 ± 0.86 b**</td>
<td>2.2 ± 1.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + GA treated (300mg/Kg bw)</td>
<td>52.4 ± 2.29 b**</td>
<td>0.49 ± 1.03 b**</td>
<td>1.1 ± 0.12 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + GA treated (400mg/Kg bw)</td>
<td>41.2 ± 1.02 b**</td>
<td>0.41 ± 0.95 b**</td>
<td>0.44 ± 1.01 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic +Glibenclamide treated (600µg/Kg bw)</td>
<td>40.2 ± 1.90 c#</td>
<td>0.49 ± 2.20 c#</td>
<td>0.48 ± 1.23 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 32  Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *G.asiatica* L. (GA) for 60 days.

I

II

III

| Level of urea in experimental animals |
| I |

| Level of uric acid in experimental animals |
| II |

| Level of creatinine in experimental animals |
| III |
Effect of *Gmelina asiatica* Linn. (GA) and their formulation on histoarchitecture of pancreas

The haematoxylin and eosin stained sections of normal rat pancreatic tissue showed intact and round or elongated islets of langerhans (Plate-la) with granulated beta cells appearing darker. Small, shrunken islets and destruction of beta cells were observed in diabetic untreated animals (Plate-Ib) GA extracts treated animals showed a profound enhancement in the size and shape of islets of langerhans. Among the drug treated animals, group of animals which received maximum dose (400mg/Kg bw) showed better granulation and rejuvenation of β – cells (Plate IVc).
Photomicrographs of Histopathological Studies

GA treated

Section of rat pancreas (x380)

a - 200mg/Kg GA treated  
b - 300mg/Kg GA treated  
c - 400mg/Kg GA treated

IS - Islets of langerhans, Gl - Granulated islet, MR - moderate regeneration.
6: 5 Effect of aqueous extract of *Sphaeranthus indicus* Linn. (SI) on drug treated and untreated diabetic animals:

**Effect of *Sphaeranthus indicus* Linn. (SI) on body weight of experimental animals**

The changes in the initial and final body weight were listed in Table - 25. Observed data indicates significant improvement in the body weight (P < 0.05 at 400mg/Kg bw) in extract treated diabetic rats with respect to diabetic control group. Group VI which received reference standard drug does not show any marked variation in body weight.
Table - 25: Effect of aqueous extract of *S. indicus* L (SI) on body weight of control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Initial body weight (g)</th>
<th>Final body weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>160.6 ± 5.20</td>
<td>175.7 ± 6.25</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>161.7 ± 4.23</td>
<td>142.5 ± 3.59 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg)</td>
<td>164.5 ± 7.68</td>
<td>172.5 ± 5.24 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg)</td>
<td>164.7 ± 4.43</td>
<td>174.5 ± 3.95 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg)</td>
<td>152.7 ± 6.08</td>
<td>170.4 ± 7.21 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>153.2 ± 8.71</td>
<td>150.4 ± 7.62 c*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

"P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 33  Effect of aqueous extract of *S. indicus* L. (SI) on body weight profile of control and treated rats.
**Effect of *Sphaeranthus indicus*, Linn (SI) on blood glucose level**

The level of blood glucose in control and experimental groups after oral administration of drug for 60 days were shown in Table – 26 & Figure 34(I). Diabetic animals showed progressive increase in blood sugar. Oral administration of SI extract, significantly (P < 0.05) reduced the blood glucose levels. At the end of the experiment, group V animals which received 400mg/ Kg bw of plant extract showed marked decrease in the blood glucose level (91.8 ± 0.54 mg/dl) compared to disease control (270.4 ± 1.24mg/dl). Reference drug – glibenclamide also showed profound decrease in blood glucose level.

**Effect of *Sphaeranthus indicus*, Linn (SI) on serum insulin level**

Figure - 34(II) and Table – 26 shows the effect of plant extract on fasting serum insulin level in diabetic untreated and diabetic treated rats. The level of serum insulin was found to be reduced in diabetes induced group II animals (4.3 ± 0.73μU/ml) compared to normoglycemic rats (6.4 ± 1.61 μU/ml). Treatment with plant extract showed marked elevation in insulin level. Group IV and V animals which received 300 (5.9 ± 2.45μU/ml) and 400 (6.8 ± 1.40μU/ml) mg/Kg bw respectively caused significant (P < 0.05) increase in serum insulin level.
Results

Effect of *Sphaeranthus indicus*. Linn (SI) on glycosylated hemoglobin (HbA,C) level

The profile of HbA,C gives the 3 months predisposition of diabetic patients. Alloxan induced hyperglycemia leads to elevated levels of HbA,C as given in Table - 26 compared to normal control. On treatment with SI extract, the level of HbA,C was seen to be reduced significantly (P < 0.05, 2.1± 2.08 %) compared to diabetic control (6.6 ± 2.41%). Among the three doses of plant extract – 200, 300 and 400 mg/kg bw, the maximum dose (400mg/Kg bw) showed profound reduction in HbA,C level. Glibenclamide causes 50 % reduction (3.1 ± 1.31%) of the HbA,C level as compared to disease control.

Effect of *Sphaeranthus indicus*. Linn (SI) on liver glycogen level

The effect of MC aqueous leaf extract on liver glycogen was represented in Figure – 34 (IV). Diabetic group of animals (Group II) showed significant (P < 0.05) decrease in liver glycogen level compared to normal animals. On treatment with plant extract, there was a significant increase in glycogen levels. Animals received 200 and 300mg/ Kg bw showed a moderate increase in glycogen level whereas group of rats received 400mg/Kg bw of plant extract showed a remarkable increase in liver glycogen levels.
Table- 26: Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of S. indicus L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Blood Sugar (mg/dl)</th>
<th>Serum insulin (μU/ml)</th>
<th>HbA1C (%)</th>
<th>Glycogen (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>86.4 ± 2.20</td>
<td>6.4 ± 1.61</td>
<td>2.6 ± 3.21</td>
<td>46.5 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>270.4 ± 1.24a*</td>
<td>4.3 ± 0.73a*</td>
<td>6.6 ± 2.41a*</td>
<td>11.7 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg)</td>
<td>206.4 ± 1.45b**</td>
<td>5.1 ± 1.25b**</td>
<td>5.7 ± 2.12b**</td>
<td>22.6 ± 2.52 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg)</td>
<td>110.7 ± 2.24b**</td>
<td>5.9 ± 2.45 b **</td>
<td>3.4 ± 2.02 b **</td>
<td>36.1 ± 1.62 b **</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg)</td>
<td>91.8 ± 0.54 b **</td>
<td>6.8 ± 1.40 b **</td>
<td>2.1 ± 2.08 b **</td>
<td>41.1 ± 1.14b **</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600μg/Kg)</td>
<td>83.4 ± 1.84c&quot;</td>
<td>5.7 ± 1.23c&quot;</td>
<td>3.1 ± 1.86c&quot;</td>
<td>41.3 ± 2.86 c&quot;</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.

_A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation_
Results

Figure - 34 Levels of blood glucose, glycosylated haemoglobin (HbA₁C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I

II

III

IV

I Level of blood glucose in experimental animals

II Level of serum insulin in experimental animals

III Level of HbA₁C in experimental animals

IV Level of Liver glycogen in experimental animals
Results

**Effect of *Sphaeranthus indicus* Linn (SI) on the activity of glycolytic and gluconeogenetic enzymes in untreated and treated rats**

Effect of aqueous extract of SI leaves on the activities of Glucokinase and The activity of glucokinase were reduced in disease control animals and resumed back to normal on treatment with SI plant aqueous extract at various doses. Fructose – 1-6 – bisphosphatase were depicted in Table - 27 and Figure - 35. Disease control animals showed significant alteration in the above enzymes. Fructose – 1-6 – bisphosphatase was found to be elevated (124.7 ± 1.23 μ moles of PO₄ liberated/ min/ mg pro) significantly when compared to normal group of animals (26.4 ± 2.61 μ moles of PO₄ liberated/ min/ mg pro). Treatment of animals with SI caused remarkable decrease in fructose – 1-6 – bisphosphatase activity. The higher dose of plant extract (400mg/ Kg bw) caused phenomenal decrease (P < 0.05) in enzyme level and in turn enzymes activity.

The activity of Glucose –6 – phosphatase and Glucose –6 – phosphate dehydrogenase on untreated and plant extract treated animals were presented graphically in Figure – 35 III & IV. Alloxan induced diabetic rats showed marked elevation (15.4 ± 1.24nmoles of Pi liberated/ min/ mg pro) in Glucose –6 – phosphatase activity, when compared to normal rats (7.7± 0.21 24nmoles of Pi liberated/ min/ mg pro). Treatment with plant extract at a dose level of 200, 300 and 400mg/ Kg bw and glibenclamide for 60 days showed significant (P < 0.05) reduction in the enzyme activity. The effect was found to be dose dependent.

SI extract showed marked variation in the activity of Glucose –6 – phosphate dehydrogenase. Diabetic rats (Group II) showed a profound decrease in enzyme activity and found to resume back to normal on administration of plant extract for 60 days. Group V animals were found to have significant (P < 0.05) increase in Glucose –6 – phosphate dehydrogenase and its effect were in par with standard drug treated (Group VI) animals.
Table – 27: Effect of aqueous extract of *S. indicus* L (SI) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Glucokinase (μ moles of Glu- 6-PO₄ formed/ min/ mg protein)</th>
<th>Fructose – 1- 6 -bisphosphatase (μ moles of PO₄ liberated/ min/ mg protein)</th>
<th>Glucose –6 –phosphatase (nmoles of Pi liberated/ min/ mg protein)</th>
<th>Glucose – 6 –phosphate dehydrogenase, (μ moles of NADPH formed/ min/ mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>136.7 ± 1.26</td>
<td>26.4 ± 2.61</td>
<td>7.7 ± 0.21</td>
<td>11.4 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>96.2 ± 2.05a*</td>
<td>124.7 ± 1.23a*</td>
<td>15.4 ± 1.24a*</td>
<td>8.9 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated</td>
<td>104.6 ± 3.01 b**</td>
<td>87.8 ± 1.76 b**</td>
<td>12.2 ± 3.04b**</td>
<td>9.8 ± 2.56 b**</td>
</tr>
<tr>
<td></td>
<td>(200mg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated</td>
<td>124.7 ± 1.54 b**</td>
<td>50.9 ± 2.32 b**</td>
<td>11.1 ± 1.25 b**</td>
<td>10.6 ± 1.28 b**</td>
</tr>
<tr>
<td></td>
<td>(300mg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated</td>
<td>135.8 ± 2.35 b**</td>
<td>28.6 ± 1.13 b**</td>
<td>8.5 ± 2.07 b**</td>
<td>11.6 ± 2.13 b**</td>
</tr>
<tr>
<td></td>
<td>(400mg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide</td>
<td>134.6 ± 1.53 c #</td>
<td>32.4 ± 2.02 c #</td>
<td>7.1 ± 2.37 c#</td>
<td>10.0 ± 2.81 c#</td>
</tr>
<tr>
<td></td>
<td>(600μg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure - 35  Effect of aqueous extract of *S. indicus* L. (SI) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

I  Activity of Glucokinase in experimental animals
II Activity of Fructose- 1- 6 - Bisphosphatase in experimental animals
III Activity of Glucose- 1- 6 - phosphate phosphatase in experimental animals
IV Activity of Glucose - 1- 6 - phosphate dehydrogenase in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 176
**Results**

**Effect of *Sphaeranthus indicus*, Linn (SI) on serum Lipid and lipoprotein levels**

Table 28 & Figure 36 depicts the serum lipid and lipoproteins levels. Diabetic animals showed elevated levels of cholesterol (141.5 ± 3.94 mg/Kg bw), TGL (168.6 ± 2.56 mg/Kg bw), PL (122.3 ± 3.81 mg/Kg bw) and FFA (110.7 ± 2.32 mg/Kg bw) compared to normal animals. Aqueous extract of SI were found to have a significant (P < 0.05) influence on the serum lipid profiles. Animals which received 400mg/Kg bw showed remarkable decrease in the serum lipid profile compared to other dose level (200 and 300mg/Kg bw) of SI extract.

Lipoprotein levels (Table 29 & Figure 37) were also found to be altered in disease control significantly (P < 0.05) compared to normal animals. The levels of LDL and VLDL which were elevated in disease control were found to be decreased in plant extract treated animals. Group V animals that received 400mg/Kg bw showed profound decrease in the levels of LDL (40.3 ± 2.52mg/dl) and VLDL (14.3 ± 1.45 mg/dl) compared to disease control (LDL - 69.3 ± 1.52mg/dl and VLDL - 30.3 ± 1.01mg/dl). HDL level in disease control animals (22.5 ± 2.04mg/dl) were decreased significantly compared to normal rats (61.5 ± 1.42mg/dl). Treatment with test drug elevated the serum HDL levels significantly and the effects were found to be dose dependent.
Table – 28: Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/dl)</th>
<th>TGL (mg/dl)</th>
<th>PL (mg/dl)</th>
<th>FFA (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>60.8 ± 2.42</td>
<td>68.4 ± 1.81</td>
<td>58.2 ± 2.14</td>
<td>44.4 ± 1.52</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>141.5 ± 3.94 a*</td>
<td>168.6 ± 2.56 a*</td>
<td>122.3 ± 3.81 a*</td>
<td>110.7 ± 2.32 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg bw)</td>
<td>120.6 ± 2.11b**</td>
<td>102.6 ± 1.07 b**</td>
<td>102.2± 1.26 b**</td>
<td>99.8 ± 2.82 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg bw)</td>
<td>122.6 ± 3.98 b**</td>
<td>83.1± 2.26 b**</td>
<td>89.7± 2.24 b**</td>
<td>72.8 ± 3.51 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic+ SI treated (400mg/Kg bw)</td>
<td>89.4± 2.52b**</td>
<td>65.7± 2.02 b**</td>
<td>59.5± 1.05 b**</td>
<td>52.5± 1.14 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>59.8 ± 1.36 c #</td>
<td>64.7 ± 2.06 c #</td>
<td>62.3± 2.19 c #</td>
<td>47.6± 2.55 c #</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 36 Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I

II

III

IV

I Level of cholesterol in experimental animals

II Level of triglyceride in experimental animals

III Level of phospholipids in experimental animals

IV Level of free fatty acids in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*

179
Table – 29: Serum lipoprotein profile of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL - Cholesterol (mg/dl)</th>
<th>LDL - Cholesterol (mg/dl)</th>
<th>VLDL - Cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>61.5 ± 1.42</td>
<td>41.5 ± 2.01</td>
<td>13.9 ± 3.14</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>22.5 ± 2.04 a*</td>
<td>69.3 ± 1.52 a*</td>
<td>30.3 ± 1.01 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg bw)</td>
<td>30.4 ± 2.22 b**</td>
<td>52.7 ± 3.04 b**</td>
<td>23.7 ± 2.53 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg bw)</td>
<td>46.8 ± 2.08 b**</td>
<td>46.8 ± 2.25 b**</td>
<td>18.2 ± 1.24 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg bw)</td>
<td>57.9 ± 2.42 b**</td>
<td>40.3 ± 2.52 b**</td>
<td>14.3 ± 1.45 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>52.5 ± 1.32 c&quot;</td>
<td>44.7 ± 2.52 c&quot;</td>
<td>17.2 ± 2.87 c&quot;</td>
</tr>
</tbody>
</table>

[HDLC - cholesterol, LDLC - cholesterol and VLDLC - cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

"P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 37  Serum lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I

- Level of HDL-cholesterol in experimental animals

II

- Level of LDL-cholesterol in experimental animals

III

- Level of VLDL-cholesterol in experimental animals
Results

Effect of *Sphaeranthus indicus*. Linn (SI) on tissue Lipid and lipoprotein levels

Effect of plant extract on tissue lipid profile was tabulated in 30 and presented graphically in Figure - 38. In severe diabetes mellitus, increased levels of tissue cholesterol, triglycerides, phospholipids and free fatty acids were seen. Whereas treating the animals with plant extract causes significant (P < 0.05) reduction in lipid profile. The effect was found to be dose dependent. Animals received 400mg/Kg bw of plant drug showed remarkable decrease in cholesterol, triglycerides, phospholipids and free fatty acid levels, which was nearer to normal values.

Tissue lipoprotein level was also altered significantly in diabetic animals. There was a profound increase in LDL and VLDL (Table - 31) levels and a notable decrease in HDL levels. The altered levels of lipoproteins were resumed to normal on treating the diabetic animals with aqueous extract at dose levels of 200, 300 and 400mg/Kg bw.
Table – 30: Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/ 100g wet tissue)</th>
<th>TGL (mg/100g wet tissue)</th>
<th>PL (mg/ 100g wet tissue)</th>
<th>FFA (mg/ 100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>78.1 ± 1.42</td>
<td>65.2 ± 2.01</td>
<td>72.8 ± 1.14</td>
<td>58.2 ± 1.32</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>172.5 ± 2.05 a*</td>
<td>152.1 ± 2.32 a*</td>
<td>132.8± 2.81 a*</td>
<td>111.2 ± 3.42 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg bw)</td>
<td>140.2 ± 2.02b**</td>
<td>115.4 ± 2.25 b**</td>
<td>120.6± 2.85 b**</td>
<td>94.3± 3.22 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg bw)</td>
<td>100.8 ± 1.72 b**</td>
<td>90.5± 1.02 b**</td>
<td>99.5± 1.32 b**</td>
<td>81.3 ± 2.17 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg bw)</td>
<td>80.4± 3.51 b**</td>
<td>64.2± 2.29 b**</td>
<td>75.6± 1.20 b**</td>
<td>60.4± 2.29 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>89.8 ± 2.72 c#</td>
<td>71.8± 2.84 c#</td>
<td>79.7± 1.52 c#</td>
<td>65.1± 1.94 c#</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

Statistical comparison:

*P < 0.05 significant when compared to normal control
**P < 0.05 significant when compared to disease control
#P < 0.05 significant when compared to Group II

*A Study On Antidiabetic Potentials Of Selecta Herbs And A Herbal Formulation*
**Results**

Figure - 38  Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I Level of cholesterol in experimental animals

II Level of triglyceride in experimental animals

III Level of phospholipids in experimental animals

IV Level of free fatty acids in experimental animals
Table - 31: Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/ 100g wet tissue)</th>
<th>LDL -Cholesterol (mg/ 100g wet tissue)</th>
<th>VLDL -Cholesterol (mg/ 100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>45.2± 2.42</td>
<td>50.6 ± 2.61</td>
<td>15.9 ± 3.24</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>20.2 ± 2.84 a*</td>
<td>79.3 ± 1.22 a*</td>
<td>35.5± 1.56 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg bw)</td>
<td>28.2 ± 2.31 b**</td>
<td>70.4 ± 2.52 b**</td>
<td>29.6 ± 2.52 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg bw)</td>
<td>34.3 ± 2.03 b**</td>
<td>61.3± 2.95 b**</td>
<td>21.3 ± 3.12 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg bw)</td>
<td>44.2± 3.12 b**</td>
<td>52.4 ± 2.42 b**</td>
<td>15.5± 1.32 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>50.2 ± 1.83 c#</td>
<td>54.7 ± 2.14 c#</td>
<td>18.0 ± 0.79 c#</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Figure - 39  Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I Level of HDL- cholesterol in experimental animals
II Level of LDL- cholesterol in experimental animals
III Level of VLDL- cholesterol in experimental animals
Results

Effect of *Sphazranthus indicus* Linn (SI) on serum protein, transaminases (SGPT and SGOT), alkaline phosphatases and bilirubin

Table 32 shows the effect of SI extract in serum liver marker enzymes – SCPT, SGOT, ALP and protein. In diabetes mellitus, due to impaired carbohydrate metabolism, alternative source of energy is utilized by the cells. Hence there was a profound decrease in serum protein level in diabetic group (Group II) of animals. On treating the diabetic animals with MC extract, the protein is spared for tissue building and hence significant ($P < 0.05$) increase in protein was seen.

Alloxan causes profound alteration in transaminases ALP and bilirubin. Treatment with plant extract causes significant ($P < 0.05$) reduction in these serum liver marker enzymes and elevation in bilirubin levels. The enzyme activities were brought back to near normal and the effect was found to be dose dependent. Hence group V animals which received 400mg/Kg bw showed profound enzyme activity compared to group III and IV animals. Glibenclamide also had influence in enzyme activity.
### Table – 32: Effect of aqueous extract of *S. indicus* L. (SI) on Serum liver markers in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/dl)</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>58.9±3.02</td>
<td>63.7±1.23</td>
<td>48.9±1.54</td>
<td>120.6±3.23</td>
<td>0.40±2.51</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>35.1±1.04a*</td>
<td>129.6±2.82 a*</td>
<td>123.7±1.39a*</td>
<td>217.8±2.41 a*</td>
<td>1.96±1.21a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated</td>
<td>40.9±1.35 **</td>
<td>110.2±2.20 b**</td>
<td>196.5±2.23 b**</td>
<td>185.2±3.21 a**</td>
<td>1.09±1.81 a**</td>
</tr>
<tr>
<td></td>
<td>(200mg/Kg bw)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated</td>
<td>46.9±2.08 b**</td>
<td>197.6±2.24 b**</td>
<td>71.7±2.04 b**</td>
<td>161.7±1.96 a**</td>
<td>0.92±3.02 a**</td>
</tr>
<tr>
<td></td>
<td>(300mg/Kg bw)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated</td>
<td>57.5±2.00 b**</td>
<td>72.3±1.74 b**</td>
<td>51.6±1.35 b**</td>
<td>132.4±2.54 a**</td>
<td>0.42±1.35 a**</td>
</tr>
<tr>
<td></td>
<td>(400mg/Kg bw)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated</td>
<td>55.7±1.65 c&quot;</td>
<td>71.7±1.68 &quot;</td>
<td>52.6±1.57 c&quot;</td>
<td>136.7±3.52 c&quot;</td>
<td>0.61±2.35&quot;</td>
</tr>
<tr>
<td></td>
<td>(600µg/Kg bw)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

[protein, transaminases (SGPT and SGOT), alkaline phosphatase (ALP), and bilirubin]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

"P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 40  Effect of aqueous extract of *S. indicus* L. (SI) on Serum liver markers in untreated and treated rats.

I  
II  
III  
IV  
V

I  Level of protein in experimental animals  
II  Activity of SGPT in experimental animals  
III  Activity of SGOT in experimental animals  
IV  Activity of ALP in experimental animals  
V  Level of bilirubin in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Effect of Sphaeranthus indicus, Linn (SI) on antioxidant status of experimental animals

Table - 33 depicts the antioxidant status of the experimental animals. Alloxan causes damage to beta cell of langerhans by producing reactive oxygen species. This oxidative damage causes significant (P < 0.05) increase in lipid peroxidation; decrease in reduced glutathione level; and decrease in activity of superoxide dismutase and catalase. On treatment with plant extract helps the diabetic animals to resume back to normal Group V animals which received 400mg/Kg bw showed increased level of GSH and decreased level of peroxides. Activity of SOD and catalase activity were also enhanced on plant drug treated animals. Glibenclamide was found to cause influence in enzymatic and non enzymatic antioxidants.
Table – 33: Effect of aqueous extract of *S. indicus* L (SI) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/100g wet tissue)</th>
<th>LPO (nM of MDA/mg protein)</th>
<th>GSH (nM/mg protein)</th>
<th>SOD (IU/mg protein)</th>
<th>Catalase (nM of hydrogen peroxide decomposed/min/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>42.5±2.22</td>
<td>10.4±2.43</td>
<td>34.7±2.03</td>
<td>21.6±1.23</td>
<td>50.4±1.21</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>31.5±2.68a*</td>
<td>25.6±1.02 a*</td>
<td>18.9±1.02a*</td>
<td>9.9±1.21 a*</td>
<td>24.6±3.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic+ SI treated (200mg/Kg bw)</td>
<td>35.4±1.24 **</td>
<td>20.4±3.12 b**</td>
<td>20.4±2.45 b**</td>
<td>11.3±1.58 a**</td>
<td>33.7±3.01 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic+ SI treated (300mg/Kg bw)</td>
<td>39.6±3.05 b**</td>
<td>15.9±2.28 b**</td>
<td>28.4±2.15 b**</td>
<td>16.6±2.52 a**</td>
<td>47.2±2.12 a**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic+ SI treated (400mg/Kg bw)</td>
<td>41.2±3.42 b**</td>
<td>11.7±1.23 b**</td>
<td>33.1±1.40b**</td>
<td>21.8±2.01 a**</td>
<td>52.6±2.14 a**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic+ Glibenclamide treated (600µg/Kg bw)</td>
<td>36.7±3.17 c*</td>
<td>14.7±2.08c*</td>
<td>29.7±1.22c*</td>
<td>16.6±2.45 c*</td>
<td>42.2±2.42 c*</td>
</tr>
</tbody>
</table>

[Superoxide dismutase (SOD), Catalase, lipid peroxidation (LPO), glutathione (GSH)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group I and Group III, IV, V

c: Group II and VI.

Herbs And A Herbal Formulation
Figure - 41 Effect of aqueous extract of *S. indicus* L. (SI) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

I

II

III

IV

V

1 Level of protein in experimental animals
2 Level of LPO in experimental animals
3 Level of GSH in experimental animals
4 Activity of SOD in experimental animals
5 Activity of catalase in experimental animals
Results

Effect of Sphaeranthus indicus. Linn (SI) on serum urea, uric acid and creatinine

Effect of SI leaf extract on serum urea, uric acid and creatinine were presented in Table 34. The levels of urea, uric acid and creatinine were found to be increased in diabetic animals (95.6 ± 2.14, 0.78 ± 2.42, and 4.0 ± 1.25 mg/dl respectively) compared to normal group of rats (38.6 ± 1.62, 0.43 ± 1.21, 0.12 ± 2.04 mg/dl). Treatment of diabetic animals with plant extract for 60 days causes significant reduction (P < 0.05) in the urea, uric acid and creatinine levels. Group V animals showed profound decrease in all the above three parameters. Group IV animals which received standard drug show mild influence in urea, uric acid and creatinine levels.
Table – 34: Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L (SI) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Urea (mg/dl)</th>
<th>Uric acid (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>38.6 ± 1.62</td>
<td>0.43 ± 1.21</td>
<td>0.12 ± 2.04</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>95.6 ± 2.14 a*</td>
<td>0.78 ± 2.42 a*</td>
<td>4.0 ± 1.25 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + SI treated (200mg/Kg bw)</td>
<td>79.4 ± 1.52 b**</td>
<td>0.60 ± 1.16 b**</td>
<td>3.7 ± 1.02 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + SI treated (300mg/Kg bw)</td>
<td>53.2 ± 2.59 b**</td>
<td>0.52 ± 1.24 b**</td>
<td>2.1 ± 1.45 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + SI treated (400mg/Kg bw)</td>
<td>39.6 ± 1.22 b**</td>
<td>0.43 ± 1.35 b**</td>
<td>0.56 ± 1.11 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>40.2 ± 1.87 c#</td>
<td>0.49 ± 2.04 c#</td>
<td>0.48 ± 2.24 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05* significant when compared to normal control

**P < 0.05** significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure 42  Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *S. indicus* L. (SI) for 60 days.

I

II

III

I  Level of urea in experimental animals
II  Level of uric acid in experimental animals
III  Level of creatinine in experimental animals
Results

Effect of *Sphaeranthus indicus* Linn (SI) and their formulation on histoarchitecture of pancreas

The haematoxylin and eosin stained sections of normal rat pancreatic tissue showed intact and round or elongated islets of langerhans (Plate-IIa) with granulated beta cells appearing darker. Small, shrunken islets and destruction of beta cells were observed in diabetic untreated animals (Plate-IIb) SI extracts treated animals showed a profound enhancement in the size and shape of islets of langerhans. Among the drug treated animals, group of animals which received maximum dose (400mg/Kg bw) showed better granulation and rejuvenation of β - cells (Plate-Vc).
Plate - V

Photomicrographs of Histopathological Studies
SI treated

Section of rat pancreas (x380)

a - 200mg/Kg SI treated  b - 300mg/Kg SI treated  c - 400mg/Kg SI treated

IS - Islets of langerhans, GI - Granulated islet, MR - moderate regeneration.
6.6 Effect of aqueous extract of Parmelia perlata. Ach (PP) on drug treated and untreated diabetic animals:

Effect of Parmelia perlata. Ach (PP) on body weight of experimental animals

Variation in the initial and final body weight in drug treated and untreated animals were presented in Table – 35 and Figure – 43. In untreated diabetic control there was a significant reduction in body weight, whereas animals treated with PP aqueous extract showed significant (p<0.05) increase in body weight. PP at a dose level of 400mg/Kg bw showed statistically significant increase in final body weight compared to disease control.
Table – 35: Effect of aqueous extract of *P. perlata* Ach. (PP) on body weight of control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Initial body weight (g)</th>
<th>Final body weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>160.6 ± 5.20</td>
<td>175.7 ± 6.25</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>161.7 ± 4.23</td>
<td>142.5 ± 3.59 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg)</td>
<td>159.2 ± 5.93</td>
<td>166.2 ± 6.12 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg)</td>
<td>151.3 ± 5.58</td>
<td>160.3 ± 5.85 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg)</td>
<td>159.5 ± 4.47</td>
<td>175.1 ± 6.25 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>159.5 ± 4.47</td>
<td>175.1 ± 6.25 b**</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)  
*P < 0.05 significant when compared to normal control  
**P < 0.05 significant when compared to disease control  
#P < 0.05 significant when compared to Group II  
Statistical comparison:  
a: Group I and Group II  
b: Group II and Group III, IV, V  
c: Group II and VI
Results

Figure - 43  Effect of aqueous extract of *P. perlata* Ach. (PP) on body weight profile of control and treated rats.
**Effect of Parmelia perlata. Ach (PP) on blood glucose level**

Effect of GA in blood glucose level was shown in Table – 36 & Figure- 44(I). Experimental inductions of diabetes mellitus caused sever hyperglycemia which was evident from the blood glucose level of group II disease control animals. Treatment with test drug showed significant (p<0.05) decrease in the blood glucose level. PP extract at a dose level of 200, 300 and 400mg/Kg bw reduced the glucose level (211.7 ± 1.26, 159.2 ± 2.02 and 101.2 ± 2.73 mg/dl respectively) compared to diabetic untreated animals. Group VI animals which received 600µg/Kg bw of glibenclamide also caused significant decrease in blood glucose level.

**Effect of Parmelia perlata. Ach (PP) on serum insulin level**

Table – 36& Figure- 44(II) shows the effect of PP on serum insulin levels in treated and untreated rats. Group II diabetic animals showed profound decrease (4.3 ± 0.73 µU/ml) in the insulin level compared to normal control (6.4 ± 1.61 µU/ml). Treatment with plant extract were found to restore the normal insulin secretion which was evident from the significant (p<0.05) increase in the serum insulin levels found in Group III (4.9 ± 1.44µU/ml), IV (5.6 ± 1.92 µU/ml) and V (6.1 ± 1.90 µU/ml) animals. Glibenclamide treated animals also showed increased level of insulin.
Results

Effect of *Parmelia perlata. Ach* (PP) on glycosylated hemoglobin (HbA1C) level

Levels of HbA1C in normal, diabetic and drug treated animals were given in Table – 36 & Figure- 44(III). The blood glucose level was elevated on alloxan induction which results in glycation of hemoglobin. Hence there was significant increase in the HbA1C levels in diabetic rats but on treatment with PP extract the levels were brought back to near normal. The effect of plant extract was found to be dose dependent. Reference drug also decreases the HbA1C levels significantly (p<0.05) compared to diabetic control.

Effect of *Parmelia perlata. Ach* (PP) on liver glucogen level

Table – 36 & Figure- 44(IV) shows the effect of plant extract on liver glycogen levels. Administration of plant extract for 60 days increases the glycogen level significantly. Animals (Group V) which received maximum dose (400mg/Kg bw) were found to have profound increase (37.5 ± 1.25mg/g tissue) in the glycogen level. Whereas disease control animals which were untreated (Group II- 11.7 ± 0.21 mg/g issue) had very low level of liver glycogen compared to normal animals (Group I - 46.5 ± 0.91 mg/g tissue).
### Results

Table 36: Levels of blood glucose, glycosylated haemoglobin (HbA₁C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Blood Sugar (mg/dl)</th>
<th>Serum insulin (µU/ml)</th>
<th>HbA₁C (%)</th>
<th>Glycogen (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>86.4 ± 2.20</td>
<td>6.4 ± 1.61</td>
<td>2.6 ± 3.21</td>
<td>46.5 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>270.4 ± 1.24a</td>
<td>4.3 ± 0.73a</td>
<td>6.6 ± 2.41a</td>
<td>11.7 ± 0.21 a</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg)</td>
<td>211.7 ± 1.26b**</td>
<td>4.9 ± 1.44b**</td>
<td>5.9 ± 1.62b**</td>
<td>22.7 ± 2.05 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg)</td>
<td>159.2 ± 2.02b**</td>
<td>5.6 ± 1.92 b**</td>
<td>4.7 ± 1.10 b**</td>
<td>28.7 ± 1.34 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg)</td>
<td>101.2 ± 2.73 b**</td>
<td>6.1 ± 1.90 b**</td>
<td>2.9 ± 1.25 b**</td>
<td>37.5 ± 1.25 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>83.4 ± 1.85c#</td>
<td>5.7 ± 1.24c#</td>
<td>3.1 ± 1.76c#</td>
<td>41.3 ± 1.05 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

- a: Group I and Group II
- b: Group II and Group III, IV, V
- c: Group II and VI.
Results

Figure - 44 Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of *P.perlata* Ach. (PP) for 60 days.

I

II

III

IV

I Level of blood glucose in experimental animals
II Level of serum insulin in experimental animals
III Level of HbA1C in experimental animals
IV Level of Liver glycogen in experimental animals
Results

Effect of Parmelia perlata, Ach (PP) on the activity of glycolytic and gluconeogenetic enzymes in untreated and treated rats

Effect of aqueous extract of SI leaves on the activities of Glucokinase and The activity of glucokinase were reduced in disease control animals and resumed back to normal on treatment with SI plant aqueous extract at various doses. Fructose − 1- 6 − bisphosphatase were depicted in Table - 37 and Figure - 45. Disease control animals showed significant alteration in the above enzymes. Fructose − 1- 6 − bisphosphatase was found to be elevated (124.7 ± 1.23 μ moles of PO₄ liberated/ min/ mg pro) significantly when compared to normal group of animals (26.4 ± 2.61 μ moles of PO₄ liberated/ min/ mg pro). Treatment of animals with GA caused remarkable decrease in fructose − 1- 6 − bisphosphatase activity. The higher dose of plant extract (400mg/ Kg bw) caused phenomenal decrease (P < 0.05) in enzyme level and in turn enzymes activity.

The activity of Glucose −6 − phosphatase and Glucose −6 − phosphate dehydrogenase on untreated and plant extract treated animals were presented graphically in Figure - 45III & IV. Alloxan induced diabetic rats showed marked elevation (15.4 ± 1.24nmole of Pi liberated/ min/ mg pro) in Glucose −6 − phosphatase activity, when compared to normal rats (7.7± 0.21 24nmole of Pi liberated/ min/ mg pro). Treatment with plant extract at a dose level of 200, 300 and 400mg/ Kg bw and glibenclamide for 60 days showed significant (P < 0.05) reduction in the enzyme activity. The effect was found to be dose dependent.

SI extract showed marked variation in the activity of Glucose −6 − phosphate dehydrogenase. Diabetic rats (Group II) showed a profound decrease in enzyme activity and found to resume back to normal on administration of plant extract for 60 days. Group V animals were found to have significant (P < 0.05) increase in Glucose −6 − phosphate dehydrogenase and its effect were in par with standard drug treated (Group VI) animals.

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation
Table – 37: Effect of aqueous extract of *P. perlata* Ach. (PP) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Glucokinase (µ moles of Glu-6-PPO₄ formed/ min/ mg protein)</th>
<th>Fructose – 1- 6 - bisphosphatase (µ moles of PO₄ liberated/ min/ mg protein)</th>
<th>Glucose –6 – phosphatase (nmoles of Pi liberated/ min/ mg protein)</th>
<th>Glucose – 6 – phosphate dehydrogenase, (µ moles of NADPH formed/ min/ mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>136.7 ± 1.26</td>
<td>26.4 ± 2.61</td>
<td>7.7± 0.21</td>
<td>11.4 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>96.2 ± 2.05a*</td>
<td>124.7 ± 1.23a*</td>
<td>15.4 ± 1.24a*</td>
<td>8.9 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg)</td>
<td>102.7 ± 1.24 b**</td>
<td>99.7 ± 1.52 b**</td>
<td>14.7 ± 2.11 b**</td>
<td>9.5 ± 2.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg)</td>
<td>114.6 ± 1.52 b**</td>
<td>64.3 ± 1.24 b**</td>
<td>12.6 ± 2.28 b**</td>
<td>10.2 ± 1.02 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg)</td>
<td>128.5 ± 1.05 b**</td>
<td>33.7 ± 1.11 b**</td>
<td>9.7 ± 1.66 b**</td>
<td>10.9 ± 1.52 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>134.6 ± 2.65 c #</td>
<td>32.4± 1.62 c #</td>
<td>7.1 ± 0.47 c#</td>
<td>10.0 ± 1.76 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 45  Effect of aqueous extract of *P.perlata* Ach. (PP) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

I  Activity of Glucokinase in experimental animals

II  Activity of Fructose- 1-6 – Bisphosphatase in experimental animals

III  Activity of Glucose- 1-6 - phosphatase in experimental animals

IV  Activity of Glucose- 1-6 – phosphate dehydrogenase in experimental animals
Results

Effect of *Parmelia perlata* Ach (PP) on serum Lipid and lipoprotein levels

Table 38 & Figure 46 depicts the serum lipid and lipoproteins levels. Diabetic animals showed elevated levels of cholesterol (141.5 ± 3.94 mg/Kg bw), TGL (168.6 ± 2.56 mg/Kg bw), PL (122.3 ± 3.81 mg/Kg bw) and FFA (110.7± 2.32 mg/Kg bw) compared to normal animals. Aqueous extract of GA were found to have a profound influence on the serum lipid profiles. Animals which received 400mg/Kg bw showed remarkable decrease in the serum lipid profile compared to other dose level (200 and 300mg/Kg bw) of SI extract.

Lipoprotein levels were also found to be altered in disease control significantly (P < 0.05) compared to normal animals. The levels of LDL and VLDL which were elevated in disease control were found to be decreased in plant extract treated animals. Group V animals that received 400mg/ Kg bw showed profound decrease in the levels of LDL (43.1 ± 1.51 mg/dl) and VLDL (14.4± 2.05 mg/dl) compared to disease control (LDL - 69.3 ± 1.52mg/dl and VLDL - 30.3 ± 1.01mg/dl). HDL level in disease control animals (22.5 ± 2.04mg/dl) were decreased significantly compared to normal rats (61.5 ± 1.42mg/dl). Treatment with test drug elevated the serum HDL levels significantly and the effects were found to be dose dependent.
## Results

Table – 38: Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/dl)</th>
<th>TGL (mg/dl)</th>
<th>PL (mg/dl)</th>
<th>FFA (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>60.8 ± 2.42</td>
<td>68.4 ± 1.81</td>
<td>58.2 ± 2.14</td>
<td>44.4 ± 1.52</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>141.5 ± 3.94 a*</td>
<td>168.6 ± 2.56 a*</td>
<td>122.3 ± 3.81 a*</td>
<td>110.7 ± 2.32 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>122.4 ± 1.25b**</td>
<td>121.7 ± 2.02 b**</td>
<td>111.6 ± 1.42 b**</td>
<td>96.5 ± 2.28 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>100.7 ± 1.32 b**</td>
<td>90.7 ± 1.36 b**</td>
<td>85.2 ± 2.04 b**</td>
<td>69.9 ± 1.21 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic+ PP treated (400mg/Kg bw)</td>
<td>79.5 ± 2.44 b**</td>
<td>72.1 ± 1.53 b**</td>
<td>61.2 ± 2.05 b**</td>
<td>48.7 ± 1.71 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>59.8 ± 3.20 c*</td>
<td>64.7 ± 2.22 c*</td>
<td>62.3 ± 1.57 c*</td>
<td>47.6 ± 1.62 c*</td>
</tr>
</tbody>
</table>

(cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA))

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation* 208
Results

Figure - 46 Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

**I**

![Graph I](image1)

**II**

![Graph II](image2)

**III**

![Graph III](image3)

**IV**

![Graph IV](image4)

I  Level of cholesterol in experimental animals
II  Level of triglyceride in experimental animals
III  Level of phospholipids in experimental animals
IV  Level of free fatty acids in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Table - 39: Serum lipoprotein profile of untreated and treated rats after oral administration of aqueous extract of *P.perlata* Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/dl)</th>
<th>LDL -Cholesterol (mg/dl)</th>
<th>VLDL -Cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>61.5 ± 1.42</td>
<td>41.5 ± 2.01</td>
<td>13.9 ± 3.14</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>22.5 ± 2.04 a*</td>
<td>69.3 ± 1.52 a*</td>
<td>30.3 ± 1.01 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>29.6 ± 1.22b**</td>
<td>55.3 ± 1.26 b**</td>
<td>24.2± 1.25 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>39.2 ± 1.62 b**</td>
<td>49.1 ± 1.23 b**</td>
<td>19.4± 2.24 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg bw)</td>
<td>50.7 ± 2.41 b**</td>
<td>43.1 ± 1.51 b**</td>
<td>14.4± 2.05 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>52.5 ± 1.38 c*</td>
<td>44.7 ± 3.54 c*</td>
<td>17.2 ± 1.37 c*</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Figure - 47  Serum lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

I  Level of HDL- cholesterol in experimental animals
II  Level of LDL- cholesterol in experimental animals
III  Level of VLDL- cholesterol in experimental animals
Effect of *Parmelia perlata, Ach* (PP) on tissue Lipid and lipoprotein levels

Effect of plant extract on tissue lipid profile was tabulated in 40 and presented graphically in Figure - 48. In severe diabetes mellitus, increased levels of tissue cholesterol, triglycerides, phospholipids and free fatty acids were seen. Whereas treating the animals with plant extract causes significant (P < 0.05) reduction in lipid profile. The effect was found to be dose dependent. Animals received 400mg/Kg bw of plant drug showed remarkable decrease in cholesterol, triglycerides, phospholipids and free fatty acid levels, which was nearer to normal values.

Tissue lipoprotein level (Table 41 & Figure 49) was also altered significantly in diabetic animals. There was a profound increase in LDL and VLDL levels and a notable decrease in HDL levels. The altered levels of lipoproteins were resumed to normal on treating the diabetic animals with aqueous extract at dose levels of 200, 300 and 400mg/Kg bw.
Table 40: Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of P.perlata Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/100g wet tissue)</th>
<th>TGL (mg/100g wet tissue)</th>
<th>PL (mg/100g wet tissue)</th>
<th>FFA (mg/100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>78.1 ± 1.42</td>
<td>65.2 ± 2.01</td>
<td>72.8 ± 1.14</td>
<td>58.2 ± 1.32</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>172.5 ± 2.05 a*</td>
<td>152.1 ± 2.32 a*</td>
<td>132.8 ± 2.81 a*</td>
<td>111.2 ± 3.42 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>142.4 ± 1.40b**</td>
<td>118.5 ± 2.06 b**</td>
<td>111.1 ± 2.06 b**</td>
<td>94.7 ± 2.36 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>99.2 ± 1.23 b**</td>
<td>89.1 ± 1.02 b**</td>
<td>96.5 ± 1.21 b**</td>
<td>67.2 ± 1.58 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg bw)</td>
<td>75.7 ± 2.58 b**</td>
<td>67.9 ± 1.25 b**</td>
<td>77.7 ± 1.28 b**</td>
<td>60.7 ± 1.46 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>89.8 ± 2.26 c#</td>
<td>71.8 ± 2.04 c#</td>
<td>79.7 ± 1.05 c#</td>
<td>65.1 ± 2.24 c#</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure - 48  Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

I

II

III

IV

- **I**  Level of cholesterol in experimental animals
- **II**  Level of triglyceride in experimental animals
- **III**  Level of phospholipids in experimental animals
- **IV**  Level of free fatty acids in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Table – 41: Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/100g wet tissue)</th>
<th>LDL -Cholesterol (mg/100g wet tissue)</th>
<th>VLDL - Cholesterol (mg/100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>45.2 ± 2.42</td>
<td>50.6 ± 2.61</td>
<td>15.9 ± 3.24</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>20.2 ± 2.84 a*</td>
<td>79.3 ± 1.22 a*</td>
<td>35.5 ± 1.56 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>28.7 ± 3.12 b**</td>
<td>66.1 ± 1.26 b**</td>
<td>31.4 ± 2.32 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>36.4 ± 2.41 b**</td>
<td>58.4 ± 1.14 b**</td>
<td>21.4 ± 1.02 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg bw)</td>
<td>47.1 ± 1.32 b**</td>
<td>52.3 ± 2.15 b**</td>
<td>16.2 ± 2.02 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>50.2 ± 1.90 c#</td>
<td>54.7 ± 3.24 c#</td>
<td>18.0 ± 1.29 c#</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*p < 0.05 significant when compared to normal control

**p < 0.05 significant when compared to disease control

*p < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure - 49  Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of \textit{P. perlata} Ach. (PP) for 60 days.

\begin{itemize}
  \item \textbf{I}  \hspace{1cm} \textbf{II}  \hspace{1cm} \textbf{III}
  \item Level of HDL- cholesterol in experimental animals
  \item Level of LDL- cholesterol in experimental animals
  \item Level of VLDL- cholesterol in experimental animals
\end{itemize}
**Results**

**Effect of *Parmelia perlata* Ach (PP) on serum protein, transaminases (SGPT and SGOT) alkaline phosphatases, and bilirubin**

Effect of plant on serum markers were presented in Table 42 & Figure 50. The serum protein were found to be decrease in disease control animals and on treatment with plant extract an glibenclamide, the level were found to be elevated significantly (P < 0.05). The activity of serum markers were altered significantly on alloxan induction (Group II). Extract of test drug decreased the levels of GPT, GOT, ALP and Bilirubin significantly. The effects were found to be dose dependent. Glibenclamide also showed moderate influence on the serum markers but not as effective as plant extract treated animals.
### Table – 42: Effect of aqueous extract of *P. perlata* Ach. (PP) on Serum liver markers in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/dl)</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>58.9±3.02</td>
<td>63.7±1.23</td>
<td>48.9±1.54</td>
<td>120.6±3.23</td>
<td>0.40±2.51</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>35.1±1.04</td>
<td>129.6±2.82</td>
<td>123.7±1.39</td>
<td>217.8±2.41</td>
<td>1.96±1.21</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>39.7±1.44</td>
<td>114.7±3.11b</td>
<td>111.5±3.01</td>
<td>209.2±1.22</td>
<td>1.21±1.23</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>44.7±1.63b</td>
<td>102.6±2.23b</td>
<td>82.3±2.24b</td>
<td>171.2±1.44</td>
<td>0.82±0.75</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg bw)</td>
<td>53.7±2.12b</td>
<td>79.3±2.05b</td>
<td>60.4±1.45b</td>
<td>139.6±2.12</td>
<td>0.46±1.81</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>55.7±2.57c #</td>
<td>71.7±1.25c #</td>
<td>52.6±0.97c #</td>
<td>136.7±4.27c #</td>
<td>0.61±1.24c #</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control
**P < 0.05 significant when compared to disease control
#P < 0.05 significant when compared to Group II

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Figure - 50  Effect of aqueous extract of *P. perlata* Ach. (PP) on Serum liver markers in untreated and treated rats.

I

II

III

IV

V

I Level of protein in experimental animals
II Activity of SGPT in experimental animals
III Activity of SGOT in experimental animals
IV Activity of ALP in experimental animals
V Level of bilirubin in experimental animals
Effect of *Parmelia perlata* Ach (PP) on antioxidant status of experimental animals

Induction of alloxan at a dose level of 150mg/Kg bw causes oxidative damage to the pancreatic beta cells. This leads to profound alterations in the antioxidant status of the diseased animals. Treatment with the plant extract at dose levels of 200, 300 and 400mg/Kg bw restored 90% of the normal antioxidant activities. The lipidperoxide levels were reduced, GSH levels were increased, activities of SOD and catalase were increased significantly (Table 43 & Figure 51). The effects of test drug on enzymatic and non enzymatic antioxidants were found to be dose dependent. Group VI animals which received standard drug, glibenclamide also maintains antioxidant status but not in par with plant extract treated animals.
Table 43: Effect of aqueous extract of *P. perlata* Ach. (PP) on tissue protein, enzymatic and non enzymatic [lipid peroxidation (LPO), glutathione (GSH)] antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/100g wet tissue)</th>
<th>LPO (nM of MDA/mg protein)</th>
<th>GSH (nM/mg protein)</th>
<th>SOD (IU/mg protein)</th>
<th>Catalase (nM of hydrogen peroxide decomposed/min/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>42.5±2.22</td>
<td>10.4±2.43</td>
<td>34.7±2.03</td>
<td>21.6±1.23</td>
<td>50.4±1.21</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>31.5 ± 2.68*</td>
<td>25.6 ±1.02a*</td>
<td>18.9±1.02a*</td>
<td>9.9±1.21a*</td>
<td>24.6±3.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic+ PP treated (200mg/Kg bw)</td>
<td>35.8 ±2.21 **</td>
<td>20.7 ±2.44b**</td>
<td>21.3±2.00 b**</td>
<td>12.7±1.25 a**</td>
<td>30.6±3.27 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic+ PP treated (300mg/Kg bw)</td>
<td>40.2 ±2.62 b**</td>
<td>16.4±1.32 b**</td>
<td>27.4±2.12 b**</td>
<td>16.7±2.21 a**</td>
<td>36.4±2.16 a**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic+ PP treated (400mg/Kg bw)</td>
<td>44.5±3.02 b**</td>
<td>12.1±1.20 b**</td>
<td>31.2±1.25 b**</td>
<td>19.7±2.51 a**</td>
<td>49.7±3.02 a**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>36.7 ±3.17 c #</td>
<td>14.7 ±2.18c #</td>
<td>29.7 ±1.45 c #</td>
<td>16.6±2.23 c #</td>
<td>42.2±2.72 c #</td>
</tr>
</tbody>
</table>

(Superoxide dismutase (SOD), Catalase, [lipid peroxidation (LPO), glutathione (GSH)])

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 51  
Effect of aqueous extract of *P.perlata* Ach. (PP) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>I</th>
<th>II</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
<th>Group V</th>
<th>Group VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>mg/100g wet tissue</td>
<td>mg/100g wet tissue</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III</th>
<th>IV</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
<th>Group V</th>
<th>Group VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>nM of MDA/mg prot</td>
<td>nM of MDA/mg prot</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>V</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
<th>Group V</th>
<th>Group VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity of catalase in experimental animals</td>
<td>Activity of catalase in experimental animals</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

1 Level of protein in experimental animals  
II Level of LPO in experimental animals  
III Level of GSH in experimental animals  
IV Activity of SOD in experimental animals  
V Activity of catalase in experimental animals
Effect of *Parmelia perlata* Ach (PP) on serum urea, uric acid and creatinine

Effect of MC leaf extract on serum urea, uric acid and creatinine were presented in Table 44 & Figure 52. The levels of urea, uric acid and creatinine were found to be increased in diabetic animals (95.6 ± 2.14, 0.78 ± 2.42, and 4.0± 1.25 mg/ dl respectively) compared to normal group of rats (38.6± 1.62, 0.43 ± 1.21, 0.12 ± 2.04 mg/ dl). Treatment of diabetic animals with plant extract for 60 days caused significant reduction (P < 0.05) in the urea, uric acid and creatinine levels. Group V animals which received 400mg/Kg bw showed profound decrease in all the above three parameters. Group IV animals which received standard drug showed mild influence in urea, uric acid and creatinine levels.
Table 44: Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *P. perlata* Ach. (PP) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Urea (mg/dl)</th>
<th>Uric acid (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>38.6 ± 1.62</td>
<td>0.43 ± 1.21</td>
<td>0.12 ± 2.04</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>95.6 ± 2.14 a*</td>
<td>0.78 ± 2.42 a*</td>
<td>4.0 ± 1.25 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PP treated (200mg/Kg bw)</td>
<td>80.5 ± 1.12 b**</td>
<td>0.64 ± 0.72 b**</td>
<td>3.6 ± 1.72 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PP treated (300mg/Kg bw)</td>
<td>64.3 ± 2.32 b**</td>
<td>0.52 ± 1.23 b**</td>
<td>2.4 ± 1.42 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + PP treated (400mg/Kg bw)</td>
<td>40.2 ± 2.10 b**</td>
<td>0.47 ± 1.56 b**</td>
<td>0.44 ± 1.51 b**</td>
</tr>
<tr>
<td>VI</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>40.2 ± 1.28 c*</td>
<td>0.49 ± 2.32 c*</td>
<td>0.48 ± 1.63 c*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure - 52  Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *P.perlata* Ach. (PP) for 60 days.

I

II

III

Level of urea in experimental animals
Level of uric acid in experimental animals
Level of creatinine in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Effect of *Parmelia perlata* Ach (PP) and their formulation on histoarchitecture of pancreas

The haematoxylin and eosin stained sections of normal rat pancreatic islets appeared intact and were round or elongated in shape (Plate- Ila) with granulated beta cells appearing darker. Small, shrunken islets and destruction of beta cells were observed in diabetic untreated animals (Plate-Ilb) PP extracts treated animals showed a profound enhancement in the size and shape of islets of langerhans. Among the drug treated animals, group of animals which received maximum dose (400mg/Kg bw) showed better regeneration and rejuvenation of $\beta$ – cells (Plate-VIc).
Photomicrographs of Histopathological Studies
PP treated

Section of rat pancreas (x380)
a - 200mg/Kg PP treated
b - 300mg/Kg PP treated
c - 400mg/Kg PP treated

IS - Islets of langerhans, GI - Granulated islet, MR - moderate regeneration.
6.7 Effect of aqueous extract of Polyherbal formulation (PHF) on drug treated and untreated diabetic animals:

**Effect of Polyherbal formulation (PHF) on body weight of experimental animals**

Variation in the initial and final body weight in drug treated and untreated animals were presented in Table – 45 and Figure – 53. In untreated diabetic control there was a significant reduction in body weight, whereas animals treated with PHF aqueous extract showed significant (p<0.05) increase in body weight. PHF at a dose level of 500mg/Kg bw showed statistically significant increase in final body weight compared to disease control.
Table 45: Effect of aqueous extract of *Poly herbal formulation* (PHF) on body weight of control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Initial body weight (g)</th>
<th>Final body weight(g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>160.6 ± 5.20</td>
<td>175.7 ± 6.25</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>161.7 ± 4.23</td>
<td>142.5 ± 3.59 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg)</td>
<td>159.3 ± 6.03</td>
<td>165.2 ± 6.78 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg)</td>
<td>154.3 ± 7.56</td>
<td>165.5 ± 5.15 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>157.6 ± 7.68</td>
<td>160.5 ± 7.25 c***</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

***P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 53  Effect of aqueous extract of *Poly herbal formulation* (PHF) on body weight profile of control and treated rats.

![Bar graph showing body weight profile of control and treated rats for different groups.](image-url)
**Results**

**Effect of Polyherbal formulation (PHF) on blood glucose level**

Effect of PHF in blood glucose level was shown in Table – 46 & Figure- 54(1). Experimental induction of diabetes mellitus caused severe hyperglycemia which was evident from the mean blood glucose level of group II disease control animals. Treatment with test drug showed significant (p<0.05) decrease in the blood glucose level. PHF extract at a dose level of 250, and 500mg/Kg bw reduced the glucose level (123.4 ± 2.01, and 89.7 ± 1.91mg/dl respectively) compared to diabetic untreated animals. Group V animals which received 600μg/Kg bw of glibenclamide also caused significant decrease in blood glucose level.

**Effect of Polyherbal formulation (PHF) on serum insulin level**

Table – 46 & Figure- 54(II) shows the effect of PHF on serum insulin levels in treated and untreated rats. Group II alloxonized animals showed profound decrease (4.3 ± 0.73 μU/ml) in the insulin level compared to normal control (6.4 ± 1.61 μU/ml). Treatment with plant extract were found to restore the normal insulin secretion which was evident from the significant (p<0.05) increase in the serum insulin levels found in Group III (5.7 ± 1.27 μU/ml) and IV (6.8 ± 1.72 μU/ml) animals. Glibenclamide treated animals also showed increased level of insulin.
Results

Effect of Polyherbal formulation (PHF) on glycosylated hemoglobin (HbA1C) level

Levels of HbA1C in normal, diabetic and drug treated animals were given in Table - 46 & Figure- 54(III). The blood glucose level was elevated on alloxan induction which results in glycation of hemoglobin. Hence there was significant increase in the HbA1C levels in diabetic rats but on treatment with PHF extract the levels were brought back to near normal. The effect of plant extract was found to be dose dependent. Reference drug also decreases the HbA1C levels significantly (p<0.05) compared to diabetic control.

Effect of Polyherbal formulation (PHF) on liver glucogen level

Table – 46 & Figure- 54(IV) shows the effect of plant extract on liver glycogen levels. Administration of plant extract for 60 days increases the glycogen level significantly. Animals (Group IV) which received maximum dose (500mg/Kg bw) were found to have profound increase (46.6 ± 1.84 mg/g tissue) in the glycogen level. Whereas disease control animals which were untreated (Group II- 11.7 ± 0.21 mg/g issue) had very low level of liver glycogen compared to normal animals (Group 1 - 46.5 ± 0.91 mg/g tissue).
Results

Table- 46: Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Blood Sugar (mg/dl)</th>
<th>Serum insulin (µU/ml)</th>
<th>HbA1C (%)</th>
<th>Glycogen (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>86.4 ± 2.20</td>
<td>6.4 ± 1.61</td>
<td>2.6 ± 3.21</td>
<td>46.5 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>270.4 ± 1.24a</td>
<td>4.3 ± 0.73a</td>
<td>6.6 ± 2.41a</td>
<td>11.7 ± 0.21a</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg)</td>
<td>123.4 ± 2.01b**</td>
<td>5.7 ± 1.27 b**</td>
<td>4.2 ± 1.58 b**</td>
<td>28.5 ± 3.01 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg)</td>
<td>82.9 ± 1.22 b**</td>
<td>6.8 ± 1.72 b **</td>
<td>1.9 ± 1.75 b **</td>
<td>46.6 ± 1.84 b **</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide (600µg/Kg)</td>
<td>83.4 ± 1.55c</td>
<td>5.7 ± 1.04c</td>
<td>3.1 ± 1.46c</td>
<td>41.3 ± 1.75 c</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 54 Levels of blood glucose, glycosylated haemoglobin (HbA1C) serum insulin and liver glycogen in control and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

I

II

III

IV

I Level of blood glucose in experimental animals
II Level of serum insulin in experimental animals
III Level of HbA1C in experimental animals
IV Level of Liver glycogen in experimental animals
Results

Effect of *Polyherbal formulation* (PHF) on the activity of glycolytic and gluconeogenetic enzymes in untreated and treated rats

Effect of aqueous extract of PHF leaves on the activities of Glucokinase and the activity of glucokinase were reduced in disease control animals and resumed back to normal on treatment with PHF plant aqueous extract at various doses. Fructose - 1-6 - bisphosphatase were depicted in Table - 47 and Figure - 55. Disease control animals showed significant alteration in the above enzymes. Fructose – 1- 6 – bisphosphatase was found to be elevated (124.7 ± 1.23 μ moles of PO₄ liberated/ min/ mg pro) significantly when compared to normal group of animals (26.4 ± 2.61 μ moles of PO₄ liberated/ min/ mg pro). Treatment of animals with PHF caused remarkable decrease in fructose – 1- 6 – bisphosphatase activity. The higher dose of plant extract (500mg/ Kg bw) caused phenomenal decrease (P < 0.05) in enzyme level and in turn enzymes activity.

The activity of Glucose –6 – phosphatase and Glucose –6 – phosphate dehydrogenase on untreated and plant extract treated animals were presented graphically in Figure - 55III & IV. Alloxan induced diabetic rats showed marked elevation (15.4 ± 1.24nmoles of Pi liberated/ min/ mg pro) in Glucose –6 – phosphatase activity, when compared to normal rats (7.7± 0.21 24nmoles of Pi liberated/ min/ mg pro). Treatment with plant extract at a dose level of 250, and 500mg/ Kg bw and glibenclamide for 60 days showed significant (P < 0.05) reduction in the enzyme activity. The effect was found to be dose dependent.

PHF extract showed marked variation in the activity of Glucose –6 – phosphate dehydrogenase. Diabetic rats (Group II) showed a profound decrease in enzyme activity and found to resume back to normal on administration of plant extract for 60 days. Group IV animals were found to have significant (P < 0.05) increase in Glucose –6 – phosphate dehydrogenase and its effect were in par with standard drug treated (Group V) animals.
Table – 47: Effect of aqueous extract of Poly herbal formulation (PHF) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Glucokinase (µ moles of Glu-6-PO₄ formed/ min/ mg protein)</th>
<th>Fructose – 1- 6- bisphosphatase (µ moles of PO₄ liberated/ min/ mg protein)</th>
<th>Glucose –6– phosphatase (nmoles of Pi liberated/ min/ mg protein)</th>
<th>Glucose – 6– phosphate dehydrogenase, (µ moles of NADPH formed/ min/ mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>136.7 ± 1.26</td>
<td>26.4 ± 2.61</td>
<td>7.7± 0.21</td>
<td>11.4 ± 0.91</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic</td>
<td>96.2 ± 2.05a*</td>
<td>124.7 ± 1.23a*</td>
<td>15.4 ± 1.24a*</td>
<td>8.9 ± 0.21 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated</td>
<td>116.8 ± 3.04 b**</td>
<td>71.6 ± 2.42 b**</td>
<td>12.5 ± 1.81b**</td>
<td>10.5 ± 2.86 b**</td>
</tr>
<tr>
<td>(250mg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated</td>
<td>132.5 ± 2.22 b**</td>
<td>28.1 ± 1.48 b**</td>
<td>8.4± 2.34 b**</td>
<td>11.5 ± 1.45 b**</td>
</tr>
<tr>
<td>(500mg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide</td>
<td>134.6 ± 2.45 c</td>
<td>32.4 ± 1.24 c</td>
<td>7.1± 1.47 c</td>
<td>10.0 ± 1.86 c</td>
</tr>
<tr>
<td>(600µg/Kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Figure - 55  Effect of aqueous extract of *Poly herbal formulation* (PHF) on the activities of glycolytic and gluconeogenic enzymes in control and treated rats.

I  Activity of Glucokinase in experimental animals

II Activity of Fructose-1-6-Bisphosphatase in experimental animals

III Activity of Glucose-1-6-phosphatase in experimental animals

IV Activity of Glucose-1-6-phosphate dehydrogenase in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Effect of Polyherbal formulation (PHF) on serum Lipid and lipoprotein levels

Table 48 & Figure 56 depicts the serum lipid and lipoproteins levels. Diabetic animals showed elevated levels of cholesterol (141.5 ± 3.94 mg/Kg bw), TGL (168.6 ± 2.56 mg/Kg bw), PL (122.3 ± 3.81 mg/Kg bw) and FFA (110.7 ± 2.32 mg/Kg bw) compared to normal animals. PHF were found to have a profound influence on the serum lipid profiles. Animals which received 500mg/Kg bw showed remarkable decrease in the serum lipid profile compared to other dose level (250 mg/Kg bw) of PHF extract.

Lipoprotein levels were also found to be altered in disease control significantly (P < 0.05) compared to normal animals. The levels of LDL and VLDL which were elevated in disease control were found to be decreased in plant extract treated animals. Group IV animals that received 500mg/ Kg bw showed profound decrease in the levels of LDL (40.1 ± 1.73 mg/dl) and VLDL (12.6 ± 2.52 mg/dl) compared to disease control (LDL - 69.3 ± 1.52mg/dl and VLDL - 30.3 ± 1.01mg/dl). HDL level in disease control animals (22.5 ± 2.04mg/dl) were decreased significantly compared to normal rats (61.5 ± 1.42mg/dl). Treatment with test drug elevated the serum HDL levels significantly and the effects were found to be dose dependent.

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation
### Table 48: Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/dl)</th>
<th>TGL (mg/dl)</th>
<th>PL (mg/dl)</th>
<th>FFA (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>60.8 ± 2.42</td>
<td>68.4 ± 1.81</td>
<td>58.2 ± 2.14</td>
<td>44.4 ± 1.52</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>141.5 ± 3.94 a*</td>
<td>168.6 ± 2.56 a*</td>
<td>122.3 ± 3.81 a*</td>
<td>110.7 ± 2.32 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>102.4 ± 1.85b**</td>
<td>112.7 ± 2.32 b**</td>
<td>91.4 ± 1.33 b**</td>
<td>76.5 ± 2.42 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>60.6 ± 1.42b**</td>
<td>62.3 ± 3.06 b**</td>
<td>57.2 ± 1.08 b**</td>
<td>45.2 ± 1.01 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>59.8 ± 3.81 c*</td>
<td>64.7 ± 2.22 c*</td>
<td>62.3 ± 1.57 c*</td>
<td>47.6 ± 1.62 c*</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

*P < 0.05 significant when compared to Group II

**P < 0.05 significant when compared to Group I and Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure 56 Serum lipid profiles of untreated and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

I Level of cholesterol in experimental animals
II Level of triglyceride in experimental animals
III Level of phospholipids in experimental animals
IV Level of free fatty acids in experimental animals

A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation
### Results

Table – 49: Serum lipoprotein profile of untreated and treated rats after oral administration of aqueous extract of *Poly herbal formulation* (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/dl)</th>
<th>LDL -Cholesterol (mg/dl)</th>
<th>VLDL -Cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>61.5 ± 1.42</td>
<td>41.5 ± 2.01</td>
<td>13.9 ± 3.14</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>22.5 ± 2.04 a*</td>
<td>69.3 ± 1.52 a*</td>
<td>30.3 ± 1.01 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>37.8 ± 1.45b**</td>
<td>49.2 ± 2.01 b**</td>
<td>21.7 ± 1.25 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>59.2 ± 2.12 b**</td>
<td>40.1 ± 1.73 b**</td>
<td>12.6 ± 2.52 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>52.5 ± 1.38 c#</td>
<td>44.7 ± 3.24 c#</td>
<td>17.2 ± 1.87 c#</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Figure - 57  Serum lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

I

II

III

I Level of HDL- cholesterol in experimental animals
II Level of LDL- cholesterol in experimental animals
III Level of VLDL- cholesterol in experimental animals
Results

Effect of Polyherbal formulation (PHF) on tissue Lipid and lipoprotein levels

Effect of plant extract on tissue lipid profile was Tabulated in 50 and presented graphically in Figure - 58. In severe diabetes mellitus, increased levels of tissue cholesterol, triglycerides, phospholipids and free fatty acids were seen. Whereas treating the animals with plant extract causes significant (P < 0.05) reduction in lipid profile. The effect was found to be dose dependent. Animals received 500mg/Kg bw of plant drug showed remarkable decrease in cholesterol, triglycerides, phospholipids and free fatty acid levels, which was nearer to normal values.

Tissue lipoprotein level was also altered significantly in diabetic animals. There was a profound increase in LDL and VLDL levels and a notable decrease in HDL levels. The altered levels of lipoproteins were resumed to normal on treating the diabetic animals with aqueous extracts at dose levels of 250 and 500mg/Kg bw.
Table – 50: Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of *Poly herbal formulation* (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Cholesterol (mg/100g wet tissue)</th>
<th>TGL (mg/100g wet tissue)</th>
<th>PL (mg/100g wet tissue)</th>
<th>FFA (mg/100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>78.1 ± 1.42</td>
<td>65.2 ± 2.01</td>
<td>72.8 ± 1.14</td>
<td>58.2 ± 1.32</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>172.5 ± 2.05 a*</td>
<td>152.1 ± 2.32 a*</td>
<td>132.8 ± 2.81 a*</td>
<td>111.2 ± 3.42 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>112.8 ± 2.20 b**</td>
<td>105.5 ± 2.00 b**</td>
<td>98.4 ± 2.26 b**</td>
<td>84.7 ± 2.48 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>79.2 ± 1.43 b**</td>
<td>64.1 ± 1.02 b**</td>
<td>70.5 ± 1.82 b**</td>
<td>58.9 ± 1.08 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>89.8 ± 2.26 c*</td>
<td>71.8 ± 2.04 c*</td>
<td>79.7 ± 1.35 c*</td>
<td>65.1 ± 2.24 c*</td>
</tr>
</tbody>
</table>

[cholesterol, triglyceride (TGL), phospholipids (PL) and free fatty acid (FFA)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

**Statistical comparison:**

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 58  Tissue lipid profiles of untreated and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

I Level of cholesterol in experimental animals
II Level of triglyceride in experimental animals
III Level of phospholipids in experimental animals
IV Level of free fatty acids in experimental animals
### Results

Table – 51: Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *Poly* herbal formulation (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>HDL -Cholesterol (mg/100g wet tissue)</th>
<th>LDL -Cholesterol (mg/100g wet tissue)</th>
<th>VLDL -Cholesterol (mg/100g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>45.2 ± 2.42</td>
<td>50.6 ± 2.61</td>
<td>15.9 ± 3.24</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>20.2 ± 2.84 a*</td>
<td>79.3 ± 1.22 a*</td>
<td>35.5 ± 1.56 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>31.5 ± 2.89 b**</td>
<td>55.1 ± 1.06 b**</td>
<td>22.4 ± 2.44 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>46.7 ± 2.71 b**</td>
<td>47.4 ± 2.18 b**</td>
<td>14.2 ± 1.52 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide treated (600µg/Kg bw)</td>
<td>50.2 ± 1.96 c*</td>
<td>54.7 ± 3.24 c*</td>
<td>18.0 ± 1.29 c*</td>
</tr>
</tbody>
</table>

[HDL - cholesterol, LDL – cholesterol and VLDL – cholesterol]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control
**P < 0.05 significant when compared to disease control

Statistical comparison:
a: Group I and Group II
b: Group II and Group III, IV, V
c: Group II and VI.
Results

Figure – 59 Tissue lipoprotein profiles of untreated and treated rats after oral administration of aqueous extract of *Poly herbal formulation* (PHF) for 60 days.

I Level of HDL- cholesterol in experimental animals
II Level of LDL- cholesterol in experimental animals
III Level of VLDL- cholesterol in experimental animals
Effect of Polyherbal formulation (PHF) on serum protein, transaminases (SGPT and SGOT), alkaline phosphatases, and Bilirubin

Effect of plant on serum markers were presented in Table 52 & Figure 60. The serum protein were found to be decrease in disease control animals and on treatment with plant extract an glibenclamide, the level were found to be elevated significantly (P < 0.05). The activity of serum markers were altered significantly on alloxan induction (Group II). Extract of test drug decreased the levels of GPT, GOT, ALP and Bilirubin significantly. The effects were found to be dose dependent. Glibenclamide also showed moderate influence on the serum markers but not as effective as plant extract treated animals.
Table – 52: Effect of aqueous extract of *Poly herbal formulation* (PHF) on Serum liver markers in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/dl)</th>
<th>SGPT (IU/dl)</th>
<th>SGOT (IU/dl)</th>
<th>ALP (IU/dl)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>58.9±3.02</td>
<td>63.7±1.23</td>
<td>48.9±1.54</td>
<td>120.6±3.23</td>
<td>0.40±2.51</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>35.1 ±1.04a*</td>
<td>129.6±2.82 a*</td>
<td>123.7±1.39a*</td>
<td>217.8±2.41a*</td>
<td>1.96±1.21a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>45.5 ±2.04 **</td>
<td>109.4±2.01 b**</td>
<td>82.3 ±2.91 b**</td>
<td>184.3±1.02 a**</td>
<td>1.01±1.00 a**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>57.4±1.13b**</td>
<td>65.2±3.30 b**</td>
<td>50.7±2.18 b**</td>
<td>124.7±1.04 a**</td>
<td>0.42±0.95 a**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Glibenclamide treated (600μg/Kg bw)</td>
<td>55.7±2.27c*</td>
<td>71.7±1.45c*</td>
<td>52.6±1.17c*</td>
<td>136.7±4.27c*</td>
<td>0.61±1.64c*</td>
</tr>
</tbody>
</table>

[protein, transaminases (SGPT and SGOT), alkaline phosphatase (ALP), and bilirubin]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Results

Figure - 60  Effect of aqueous extract of *Poly herbal formulation* (PHF) on Serum liver markers in untreated and treated rats.

I

Group I \ Group II \ Group III
Group IV \ Group V

II

Group I \ Group II \ Group III
Group IV \ Group V

III

Group I \ Group II \ Group III
Group IV \ Group V

IV

Group I \ Group II \ Group III
Group IV \ Group V

V

Group I \ Group II \ Group III
Group IV \ Group V

I  Level of protein in experimental animals
II  Activity of SGPT in experimental animals
III Activity of SGOT in experimental animals
IV  Activity of ALP in experimental animals
V  Level of bilirubin in experimental animals

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Effect of Polyherbal formulation (PHF) on antioxidant status of experimental animals

Induction of alloxan at a dose level of 150mg/Kg bw causes oxidative damage to the pancreatic beta cells. This leads to profound alterations in the antioxidant status of the diseased animals. Treatment with the plant extract at dose levels of 200, 300 and 400mg/Kg bw restored the normal antioxidant activities. The lipidperoxide levels were reduced, GSH levels were increased, activities of SOD and catalase were increased significantly (Table 53 & figure 61). The effects of test drug on enzymatic and non enzymatic antioxidants were found to be dose dependent. Group VI animals which received standard drug, glibenclamide also maintains antioxidant status but not in par with PHF treated animals.
Table – 53: Effect of aqueous extract of *Poly herbal formulation* (PHF) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Protein (mg/100g wet tissue)</th>
<th>LPO (nM of MDA/mg protein)</th>
<th>GSH (nM/mg protein)</th>
<th>SOD (IU/mg protein)</th>
<th>Catalase (nM of hydrogen peroxide decomposed/min/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>42.5±2.22</td>
<td>10.4±2.43</td>
<td>34.7±2.03</td>
<td>21.6±1.23</td>
<td>50.4±1.21</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>31.5±2.68a°</td>
<td>25.6±1.02 a°</td>
<td>18.9±1.02a°</td>
<td>9.9±1.21a°</td>
<td>24.6±3.21a°</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>35.8±2.21 °°</td>
<td>17.2±2.04 b°°</td>
<td>26.1±2.58 b°°</td>
<td>14.2±1.45 a°°</td>
<td>36.4±3.04 a°°</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>40.2±2.62 b°°</td>
<td>10.2±1.72 b°°</td>
<td>35.1±2.48 b°°</td>
<td>20.7±2.11 a°°</td>
<td>49.7±2.52 a°°</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic + Gilbencalamide treated (600μg/Kg bw)</td>
<td>36.7±3.17 c°°</td>
<td>14.7±2.68c°°</td>
<td>29.7±1.55 c°°</td>
<td>16.6±2.28 c°°</td>
<td>42.2±2.12 c°°</td>
</tr>
</tbody>
</table>

[Superoxide dismutase (SOD), Catalase, lipid peroxidation (LPO), glutathione (GSH)]

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.

*A Study On Antidiabetic Potentials Of Selected Herbs And A Herbal Formulation*
Results

Figure - 61  Effect of aqueous extract of *Poly herbal formulation* (PHF) on tissue protein, enzymatic and non enzymatic antioxidants in untreated and treated rats.

I Level of protein in experimental animals
II Level of LPO in experimental animals
III Level of GSH in experimental animals
IV Activity of SOD in experimental animals
V Activity of catalase in experimental animals
Results

**Effect of Polyherbal formulation (PHF) on serum urea, uric acid and creatinine**

Effect of MC leaf extract on serum urea, uric acid and creatinine were presented in Table 54 & Figure 62. The levels of urea, uric acid and creatinine were found to be increased in diabetic animals (95.6 ± 2.14, 0.78 ± 2.42, and 4.0± 1.25 mg/ dl respectively) compared to normal group of rats (38.6± 1.62, 0.43 ± 1.21, 0.12 ± 2.04 mg/ dl). Treatment of diabetic animals with plant extract for 60 days caused significant reduction (P < 0.05) in the urea, uric acid and creatinine levels. Group V animals which received 500mg/Kg bw showed profound decrease in all the above three parameters. Group V animals which received standard drug show mild influence in urea, uric acid and creatinine levels.
Table – 54: Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of Poly herbal formulation (PHF) for 60 days.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Parameters</th>
<th>Urea (mg/dl)</th>
<th>Uric acid (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal control</td>
<td>38.6± 1.62</td>
<td>0.43 ± 1.21</td>
<td>0.12 ± 2.04</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control</td>
<td>95.6 ± 2.14 a*</td>
<td>0.78 ± 2.42 a*</td>
<td>4.0± 1.25 a*</td>
</tr>
<tr>
<td>III</td>
<td>Diabetic + PHF treated (250mg/Kg bw)</td>
<td>66.7 ± 2.22b**</td>
<td>0.59 ± 1.02 b**</td>
<td>2.5± 1.42 b**</td>
</tr>
<tr>
<td>IV</td>
<td>Diabetic + PHF treated (500mg/Kg bw)</td>
<td>39.2 ± 2.32 b**</td>
<td>0.41± 1.49 b**</td>
<td>0.14 ± 1.70 b**</td>
</tr>
<tr>
<td>V</td>
<td>Diabetic +Glibenclamide treated (600μg/Kg bw)</td>
<td>40.2 ± 1.58 c#</td>
<td>0.49 ± 2.82 c#</td>
<td>0.48 ± 1.63 c#</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E (n = 6)

*P < 0.05 significant when compared to normal control

**P < 0.05 significant when compared to disease control

#P < 0.05 significant when compared to Group II

Statistical comparison:

a: Group I and Group II

b: Group II and Group III, IV, V

c: Group II and VI.
Figure - 62  Serum urea, uric acid and creatinine levels of untreated and treated rats after oral administration of aqueous extract of *Poly herbal formulation* (PHF) for 60 days.

I Level of urea in experimental animals
II Level of uric acid in experimental animals
III Level of creatinine in experimental animals
Results

Effect of Polyherbal formulation (PHF) and their formulation on histoarchitecture of pancreas

The haematoxylin and eosin stained sections of normal rat pancreatic tissue showed intact and round or elongated islets of langerhans (Plate-IIa) with granulated beta cells appearing darker. Small, shrunken islets and destruction of beta cells were observed in diabetic untreated animals (Plate-IIb) PHF extracts treated animals showed a profound enhancement in the size and shape of islets of langerhans. Among the drug treated animals, group of animals which received maximum dose (400mg/kg bw) showed better granulation and rejuvenation of β-cells (Plate-VIIc).
Photomicrographs of Histopathological Studies

PHF treated

Section of rat pancreas (x380)

a - 200mg/Kg PHF treated  b - 300mg/Kg PHF treated  c - 400mg/Kg PHF treated

IS - Islets of langerhans, GI - Granulated islet, MR - moderate regeneration.