HYPONIDDD TRIAL FOR PCOS

Hyponidd an Indian herbomineral preparation is equally efficacious and better tolerated than Metformin in patients with polycystic ovarian syndrome

Published at European Society of Human Reproduction & Embryology’s 22nd Annual Meeting at Prague; 2006

A randomized and comparative study was conducted by Dr. G. Allahbadia and team at Rotunda IVF centre, Mumbai.

One hundred and twenty three infertile PCOS women were selected and were divided in 2 groups:
- Group A – (n=73) were administered 1000 mg of metformin daily in two divided doses for six months
- Group B – (n= 50) were administered two tablets of Hyponidd twice daily for a period of six months

Hyponidd tablet was equally efficacious with better tolerability and better patient compliance than Metformin.

Eleven patients in the Hyponidd group and 16 patients in the Metformin group conceived with the help of various assisted reproductive technology (ART)

Published at European Society of Human Reproduction & Embryology’s 22nd Annual Meeting at Prague; 2006
Efficacy of herbal formulation (Hyponidd) in the management of anovulatory PCOS in women: a comparison with Metformin

(Conducted by Dr. Meena Chimote....Data on file)

An open, randomized, comparative trial was conducted in 172 patients by Dr. Meena Chimote and team at Vaunshdhara Assisted Conception Centre, Nagpur.

This 3-month trial compared the efficacy of Hyponidd (2 b.i.d.) with metformin (750 mg b.i.d).

The results indicated that although both the treatments were equally effective in countering insulin resistance, the group treated with Hyponidd lowered insulin resistance and hyperandrogenism more effectively without any side effects as compared to metformin.

These findings in the present study suggest that HYPONIDD which contains Gymnema sylvestre and Pterocarpus marsupium effectively reduce insulin resistance, LH: FSH ratio, hyperandrogenemia, acne and hirsutism equally like metformin does. However, the reduction in BMI and waist girth as well as decrease in the incidence of hyperandrogenemia with HYPONIDD treatment was more noticeable as compared to metformin treatment.

Evaluation of HYPONIDD compared to Metformin in PCOS

(Conducted by Dr. Sandhya Chhasatia....Data on file)

51 Patients with infertility for 2 - 3 years and between the age of 20 -30 years who were non diabetics and non responders to CC (Clomiphene citrate) were selected. These 51 patients were divided in to two groups. 25 patients were treated with Metformin (500 mg tds) while 26 patients were treated with HYPNIDD (2 tablets B.D.) for a period of 6 months. It was evident from the study that Metformin & Hyponidd are equally effective in insulin resistant obese PCOS patients. HYPONIDD makes ovaries more responsive to CC and Gonadotrophins also Hyponidd has less side effects than that of Metformin.
HYPONIDD TRIAL FOR DIABETES MELLITUS

Role of herbal formulation (HYPONIDD) in freshly detected type 2 Diabetes Mellitus

(Published in The Asian Journal Of Diabetology, Jan-Mar 2001 conducted by S. Chalkhor and S. Pendsey)

A double blind placebo controls study was conducted on 40 patients freshly detected with Type 2 diabetes mellitus at Diabetes clinic and Research Centre.
The study was conducted to evaluate the efficacy and safety of Hyponidd compared with placebo in freshly detected type 2 Diabetes Mellitus. 40 patients were divided into two groups:
Group A – fix dose of 2 tablets b.i.d. Hyponidd tablets for 3 months
Group B - fix dose of 2 tablets b.i.d. Placebo tablets for 3 months

Results

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Hyponidd</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 days</td>
<td>90 days</td>
</tr>
<tr>
<td>FBG (mg/dl)</td>
<td>135.57+33.53</td>
<td>102.89+24.18</td>
</tr>
<tr>
<td>PPBG (mg/dl)</td>
<td>215.84+35.62</td>
<td>163.57+32.96</td>
</tr>
<tr>
<td>GHbA (%)</td>
<td>9.70+0.498</td>
<td>9.22+0.54</td>
</tr>
<tr>
<td>Fructosamine (μmol/l)</td>
<td>320.57+25.80</td>
<td>285.31+23.72</td>
</tr>
<tr>
<td>Cholesterol (mg%)</td>
<td>195.78+44.94</td>
<td>181.73+36.86</td>
</tr>
<tr>
<td>Triglyceride (mg%)</td>
<td>177.57+61.95</td>
<td>148.31+63.17</td>
</tr>
</tbody>
</table>

Conclusion
Hyponidd is a safe drug and effective oral agent in the management of a symptomatic freshly detected type 2 diabetic patients.

Evaluation of Efficacy and Tolerability of Herbomineral Formulation (Hyponidd) in combination with Antidiabetic Agents (ADAs) in the Management of Type 2 Diabetes Mellitus in Indian patient

(Published in JAMA India – The Physicians' Update 2001, Vol. 4, No. 12…conducted by VP Pandey, Prasan Bhandari)

A double-blind placebo controlled study was conducted on 90 NIDDM patients with persistent poor glycemic control despite maximal doses of sulfonylurea and Metformin. The objective of the study was to determine the efficacy of HYPONIDD, compared with placebo, on the metabolic control of NIDDM patients inadequately controlled on maximal doses of conventional oral agents.

These 90 were randomly assigned to receive additional treatment with HYPONIDD 2 tablets three times or placebo for 12 weeks.

After 6 weeks of dietary reinforcement, efficacy was assessed by changes in HbA1c, fasting and 1-h postprandial plasma glucose and fasting lipid levels.

Results: HYPONIDD treatment was associated with significantly greater reductions in

- HbA1c (8.31 ± 1.51% to 7.01 ± 1.37%)
- Fasting plasma glucose (199.63 ± 42.13 mg/dl to 101.4 ± 16.5 mg/dl)
- 2-h postprandial glucose (306.83 ± 51.78 mg/dl to 175.10 ± 26.22 mg/dl)
- Serum cholesterol levels decreased from 271.58 ± 47.96 mg/dl to 230.95 ± 37.84 mg/dl
- Serum Triglycerides decreased from 208.13 ± 69.98 mg/dl to 160.33 ± 42.94 mg/dl
- Serum HDL increased from 44.52 ± 20.96 mg/dl to 58.62 ± 32.40 mg/dl
- Serum LDL decreased from 151.06 ± 43.73 mg/dl to 135.45 ± 33.83 mg/dl
- There were no significant adverse effects after HYPONIDD therapy

Conclusion: In NIDDM patients inadequately controlled on conventional oral agents, HYPONIDD in moderate doses resulted in beneficial effects on glycemic control, and lipid level. Additional use of HYPONIDD can be considered as a useful alternative in such patients, if they are reluctant to accept insulin therapy.

Published in JAMA India – The Physicians' Update 2001, Vol. 4, No. 12
Evaluation of Efficacy of a Herbomineral Formulation (Hyponidd) in combination with Oral Hypoglycemic Agents (OHA’s) in the management of type 2 diabetes Indian patients

Published in Medicine Update 2001 10/01… conducted by K.M. Prasanna Kumar, Mala Dharmalingam, Prasan Bhandari

Open prospective trial was conducted at M S Ramiah Medical College Hospital by K.M. Prasanna Kumar, Mala Dharmalingam, Prasan Bhandari to evaluate the efficacy of a Herbomineral Formulation (Hyponidd) in combination with Oral Hypoglycemic Agents (OHA’s) in the management of type 2 diabetes Indian patients.

35 patients with type 2 diabetes were selected and were given a dosage of Hyponidd two tablets twice daily ½ an hour before meal upto 3 months

Result:

<table>
<thead>
<tr>
<th></th>
<th>Mean Fasting Blood sugar</th>
<th>PP Blood sugar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>197.22 + 37.25</td>
<td>277.28 + 45.06</td>
</tr>
<tr>
<td>60 days</td>
<td>176.46 + 36.78</td>
<td>229.74 + 38.95</td>
</tr>
<tr>
<td>90 days</td>
<td>145.12 + 29.81</td>
<td>240.63 + 58.26</td>
</tr>
</tbody>
</table>

Conclusion
The Herbo-mineral formulation (Hyponidd) represents a safe and effective third Oral Hypoglycemic Agent that can be added in patients inadequately controlled by sulphonylurea and metformin to postpone insulin therapy.

Published in Medicine Update 2001 10/01 pp.81-84
Antihyperglycaemic and antioxidant effect of HYPONIDD, an ayurvedicherbomineral formulation in streptozotocin-induced diabetic rats

Published in *Journal of Pharmacy and Pharmacology* 2004, 56 … conducted by P. Subash Babu and P. Stanely Mainzen Prince

In the experiment, a total of 36 rats (12 normal; 24 STZ-diabeticsurviving rats) were used. The rats were divided into 6 groups of 6 rats each

- Group I, normal untreated rats
- Group II, normal rats treated with Hyponidd (200 mg/kg) orally daily for 45 days;
- Group III, STZ-treated diabetic rats;
- Group IV & V, STZ-treated diabetic rats administered Hyponidd orally (100 mg/kg and 200 mg/kg, respectively) daily for 45 days;
- Group VI, STZ-treated diabetic rats given glibenclamide orally (600µg/kg) daily for 45 days.

After 45 days of treatment, the rats were decapitated after an overnight fast. Blood was collected in heparinized tubes and plasma was separated after centrifugation. Liver tissues were excised immediately and stored in ice-cold containers.

**Results:**
Conclusion:
This study shows that oral administration of Hyponidd has antihyperglycaemic and antioxidant effects in streptozotocin-induced diabetic rats. The antioxidant phytochemicals present in the various plant constituents of Hyponidd scavenge free radicals and prevent the depletion of endogenous antioxidants. An increase in insulin levels
improves the blood glucose levels and thus Hyponidd also exhibits antihyperglycaemic activity.

Published in Journal of Pharmacy and Pharmacology 2004, 56
Antihyperlipidemic and Antioxidant Effect of HYPONIDDD in the Brain of Streptozotocin Induced Diabetic Rat

Published in *International Journal of Biological Chemistry* 1(4): 196-204, 2007... conducted by P. Subash-Babu and S. Ignacimuthu

In the experiment, a total of 42 rats (12 normal, 30 STZ-diabetic surviving rats) were used and divided into 7 groups (six in each)

- Group 1: Normal untreated rats;
- Group 2: Normal rats treated with Hyponidd (200 mg/kg b.wt.) for 45 days
- Group 3: Streptozotocin treated diabetic control rats;
- Groups 4: STZ treated diabetic rats administered with Hyponidd (50 mg mg/kg b.wt.) for 45 days
- Group 5: STZ treated diabetic rats administered with Hyponidd (100 mg/kg b.wt.) for 45 days
- Group 6: STZ treated diabetic rats administered with Hyponidd (200 mg/kg b.wt.) for 45 days
- Group 7: STZ treated diabetic rats given glibenclamide orally (600 μg/kg b.wt.) for 45 days

After 45 days of treatment, the rats were decapitated after an overnight fast. Blood was collected in heparinized tubes and plasma was separated after centrifugation. Brain tissues were excised immediately and stored in ice-cold containers.

Results:

### Table 3: Effect of hyponidd on TBARS, HP, SOD, CAT, GPX and GSH levels in normal and diabetic rat brain

<table>
<thead>
<tr>
<th>Groups</th>
<th>TBARS</th>
<th>HP</th>
<th>SOD</th>
<th>CAT</th>
<th>GSH</th>
<th>Gpx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1.58±0.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>62.38±3.55&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.96±0.69&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.93±0.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.82±0.53&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.91±0.47&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Normal + hyponidd (200 mg kg&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>1.55±0.12&lt;sup&gt;a&lt;/sup&gt;</td>
<td>62.17±3.10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.24±0.61&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.05±0.34&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10.28±0.81&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.83±0.51&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>2.89±0.18&lt;sup&gt;b&lt;/sup&gt;</td>
<td>98.83±4.16&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.28±0.27&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.83±0.05&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.72±0.36&lt;sup&gt;c&lt;/sup&gt;</td>
<td>2.90±0.15&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic + hyponidd (200 mg kg&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>1.84±0.10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.59±2.48&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.98±0.43&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.92±0.19&lt;sup&gt;d&lt;/sup&gt;</td>
<td>8.46±0.47&lt;sup&gt;d&lt;/sup&gt;</td>
<td>3.87±0.22&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic + hyponidd (100 mg kg&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>1.62±0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>67.44±1.82&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.72±0.48&lt;sup&gt;d&lt;/sup&gt;</td>
<td>2.35±0.21&lt;sup&gt;d&lt;/sup&gt;</td>
<td>9.93±0.39&lt;sup&gt;d&lt;/sup&gt;</td>
<td>3.75±0.37&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Diabetic + glibenclamide (900 μg kg&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>1.67±0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>69.38±1.08&lt;sup&gt;c&lt;/sup&gt;</td>
<td>6.21±0.47&lt;sup&gt;d&lt;/sup&gt;</td>
<td>2.12±0.20&lt;sup&gt;d&lt;/sup&gt;</td>
<td>8.89±0.28&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.81±0.33&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

**UNITS =** (TBARS - nmoles/100 g tissue; Hydroperoxides- n moles/100 g tissue; GSH-mmoles g<sup>-1</sup> wet tissue; GPX-μg of GSH consumed min<sup>-1</sup> mg<sup>-1</sup> protein; CAT-μmoles of H<sub>2</sub>O<sub>2</sub> consumed min<sup>-1</sup> mg<sup>-1</sup> protein; SOD-One unit is defined as the enzyme concentration required to inhibit the O.D at 560 nm of chromogen production by 50% in one min<sup>-1</sup> mg<sup>-1</sup> protein) Each value is mean±SD for 6 rats in each group, Values not sharing a common superscript differ significantly at p<0.05 (DMRT)
Antihyperlipidemic Effect

Table 4: Effect of hyponidd on total cholesterol, triglyceride and free fatty acids levels in normal and diabetic rat brain

<table>
<thead>
<tr>
<th>Groups</th>
<th>Total cholesterol (mg g⁻¹ wet tissue)</th>
<th>Triglyceride (mg g⁻¹ wet tissue)</th>
<th>Free fatty acids (mg g⁻¹ wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>12.89±0.82a</td>
<td>1.89±0.08b</td>
<td>1.32±0.15a</td>
</tr>
<tr>
<td>Normal + hyponidd (200 mg kg⁻¹)</td>
<td>13.47±1.29p</td>
<td>1.94±1.14g</td>
<td>1.64±0.29a</td>
</tr>
<tr>
<td>Diabetic control</td>
<td>23.89±0.81b</td>
<td>3.64±0.20f</td>
<td>2.93±0.20b</td>
</tr>
<tr>
<td>Diabetic + hyponidd (100 mg kg⁻¹)</td>
<td>16.68±1.13c</td>
<td>2.49±0.17d</td>
<td>2.01±0.07c</td>
</tr>
<tr>
<td>Diabetic + hyponidd (200 mg kg⁻¹)</td>
<td>13.29±1.04d</td>
<td>2.04±0.07e</td>
<td>1.47±0.09d</td>
</tr>
<tr>
<td>Diabetic + glibenclamide (600 µg kg⁻¹)</td>
<td>14.53±1.04e</td>
<td>2.27±0.13f</td>
<td>1.58±0.07d</td>
</tr>
</tbody>
</table>

Each value is mean±SD for 6 rats in each group, Values not sharing a common superscript differ significantly at p<0.05 (DMRT).

Conclusion:
In the present study, Hyponidd significantly restored the antioxidant enzymes and lipid profiles in STZ-induced diabetic rat brain.

Published in International Journal of Biological Chemistry 1(4): 196-204, 2007
In the experiment, a total of 42 rats (12 normal, 30 STZ-diabetic surviving rats) were used and divided into 7 groups (six in each)

- Group 1: Normal untreated rats;
- Group 2: Normal rats treated with Hyponidd (200 mg/kg b.wt.) for 45 days
- Group 3: Streptozotocin treated diabetic control rats;
- Groups 4: STZ treated diabetic rats administered with Hyponidd (50 mg/kg b.wt.) for 45 days
- Group 5: STZ treated diabetic rats administered with Hyponidd (100 mg/kg b.wt.) for 45 days
- Group 6: STZ treated diabetic rats administered with Hyponidd (200 mg/kg b.wt.) for 45 days
- Group 7: STZ treated diabetic rats given glibenclamide orally (600 μg/kg b.wt.) for 45 days

After 45 days of treatment, the rats were decapitated after an overnight fast. Blood was collected in heparinized tubes and plasma was separated after centrifugation. Liver tissues were excised immediately and stored in ice-cold containers.

Results:

### Carbohydrate Metabolism

<table>
<thead>
<tr>
<th>Groups</th>
<th>Normal</th>
<th>Normal+ Hyponidd (200 mg kg⁻¹)</th>
<th>Diabetic control</th>
<th>Diabetic+ Hyponidd (50 mg kg⁻¹)</th>
<th>Diabetic+ Hyponidd (200 mg kg⁻¹)</th>
<th>Diabetic+ glibenclamide (600 mg kg⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Hexokinase</td>
<td>0.14±0.005&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.17±0.007&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.05±0.003&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.08±0.004&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.12±0.005&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.10±0.004&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Glucose-6-phosphatase</td>
<td>0.24±0.05&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.26±0.02&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.48±0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.30±0.02&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.27±0.01&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.29±0.02&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fructose-1,6-bisphosphatase</td>
<td>0.34±0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.35±0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.71±0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.46±0.02&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.39±0.02&lt;sup&gt;ed&lt;/sup&gt;</td>
<td>0.42±0.03&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>Liver Hexokinase</td>
<td>116.48±5.26&lt;sup&gt;a&lt;/sup&gt;</td>
<td>122.80±4.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>61.81±2.39&lt;sup&gt;a&lt;/sup&gt;</td>
<td>72.67±2.64&lt;sup&lt;d&lt;/sup&gt;</td>
<td>96.52±4.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>91.30±4.59&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Glucose-6-phosphatase</td>
<td>15.28±0.98&lt;sup&gt;a&lt;/sup&gt;</td>
<td>13.75±0.92&lt;sup&gt;a&lt;/sup&gt;</td>
<td>34.41±2.34&lt;sup&lt;b&lt;/sup&gt;</td>
<td>23.50±1.50&lt;sup&gt;d&lt;/sup&gt;</td>
<td>18.03±1.01&lt;sup&gt;d&lt;/sup&gt;</td>
<td>19.01±0.71&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fructose-1,6-bisphosphatase</td>
<td>6.92±0.41&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.82±0.68&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.36±0.89&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.82±0.71&lt;sup&gt;d&lt;/sup&gt;</td>
<td>8.91±0.51&lt;sup&gt;d&lt;/sup&gt;</td>
<td>9.28±0.84&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Kidney Hexokinase</td>
<td>98.46±5.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>102.50±4.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>62.80±2.94&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.52±3.67&lt;sup&gt;d&lt;/sup&gt;</td>
<td>96.61±4.50&lt;sup&gt;a&lt;/sup&gt;</td>
<td>94.32±4.73&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Glucose-6-phosphatase</td>
<td>11.53±0.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.28±1.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24.28±1.44&lt;sup&gt;b&lt;/sup&gt;</td>
<td>18.03±0.84&lt;sup&gt;d&lt;/sup&gt;</td>
<td>12.43±0.47&lt;sup&gt;d&lt;/sup&gt;</td>
<td>14.51±0.82&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fructose-1,6-bisphosphatase</td>
<td>10.70±0.62&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.58±0.75&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24.69±1.82&lt;sup&gt;a&lt;/sup&gt;</td>
<td>18.94±0.91&lt;sup&gt;d&lt;/sup&gt;</td>
<td>11.08±0.71&lt;sup&gt;d&lt;/sup&gt;</td>
<td>12.07±0.48&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Units: Hexokinase: mmole of glucose phosphorylated/hr/mg protein. Glucose-6-phosphatase: μmole of Pi liberated/min/mg protein. Fructose-1, 6-bisphosphatase: μmole of Pi liberated/min/mg protein. Each value is Mean±SD for 6 rats in each group. Values not sharing a common superscript differ significantly at p<0.05 (DMRT)
Conclusion: In the present study, Hyponidd significantly restored the altered carbohydrate metabolizing enzymes and lipid enzymes in STZ diabetic rats.

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