International Journal of Phytopharmacology

www.onlineijp.com

e- ISSN 0975 – 9328
Print ISSN 2229 – 7472

Research article

# EVALUATION OF ANTI-DIABETIC ACTIVITY OF HYDRO ALCOHOLIC EXTRACT OF SPROUTS OF SEEDS OF *VIGNA RADIATA* ON STREPTOZOCIN INDUCED DIABETES IN WISTAR RATS

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**ABSTRACT**

Diabetes mellitus is a metabolic disorder of multiple etiologies characterized by chronic hyper glycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both[1]. Type I diabetes is due to cell destruction, usually leading to absolute insulin deficiency, Type II diabetes predominantly due to insulin resistance with relative insulin deficiency. In 2000, an estimated 171 million people in the world had diabetes and this is projected to increase to 366 million by 2030. The World Health Organization (WHO) has estimated that for some 3.4 billion people in the developing world, plants represent the primary source of medicines. Many plants are used to treat Diabetes mellitus. The mung bean *Vigna radiata* (L.) is a legume cultivated for its edible seeds and sprouts across Asia, belongs to Fabaceae family. *Vigna radiate* used for paralysis, rheumatism, coughs, fevers and liver ailments. The evaluation of Anti-diabetic activity of *Vigna radiate* was performed by following procedure. The acute oral toxicity was performed as per OECD 423 guidelines. Wistar albino rats were divided into five groups of six animals each. Group I - normal, Group II - STZ (55 mg/kg b.w., i.p) induced diabetic animals. Group III - STZ (55 mg/kg b.w., i.p) induced diabetic animals treated with Glibenclamide 5mg/kg b.w/p.o. Group IV-STZ (55 mg/kg b.w., i.p) induced diabetic animals treated with HAEVR (Hydro alcoholic extract of *Vigna radiata*) 200mg/kg b.w/ p.o. Group V - STZ (55 mg/kg b.w., i.p) induced diabetic animals were treated with HAEVR 400mg/kg b w/p.o for 28 days. On 28$^{th}$ day all animals were sacrificed, blood was collected, serum separated. Blood glucose, total cholesterol, HDL, LDL, VLDL, triglycerides, total protein, creatinine, urea, SGOT and SGPT were estimated. Histopathology of pancreas, kidney, and liver were carried out.

**Key words:** Diabetic, STZ induced diabetic, Hydro alcoholic extract of *Vigna radiate*.

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## INTRODUCTION

Plants had been used for medicinal purposes long before recorded history. The worldwide prevalence of diabetes mellitus (DM) has risen dramatically over the past two decades; based on current trends, more than 360 million individuals will have diabetes by year 2030. In addition to oral agents and insulin therapy, phytotherapy is an alternative that offers a wide range of natural resources with hypoglycaemic effects some plants provide materials recommended for people with diabetes. Herbal remedies are beneficial to patients with type 2 diabetes and they can help glucose homeostasis, but they cannot replace insulin and oral medication

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|---|---|
| DOI: http://dx.doi.org/10.21276/ijp.2017.8.2.4 | |

| Received:04.02.17 | Revised:10.03.17 | Accepted:19.03.17 |
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(WHO, 1999). Mung bean seed used for paralysis, coughs, fevers, a good source of minerals, proteins, provitamin A and vitamin B complex. In Veterinary medicine, seed paste mixed with turmeric powder applied to treat dislocated bone of cattle (Lambrides CJ & Godwin I, 2007)**.** In the present study, anti-diabetic activity of Hydro alcoholic extracts of sprout of seeds of *Vigna radiate* have been evaluated.

**Experimental Animals**

Adult wistar rats of weighing 180-220 gms were used for this study. The inbred animals were procured from the animal house of C.L. Baid Metha College of Pharmacy, Thorapakkam, Chennai - 97. They were housed five per cage under standard laboratory conditions at a room temperature at $22\pm2^0$ C with 12 hr light/dark cycle for 7 days. The animals were acclimatized to laboratory conditions for one week and provided with standard pellet chow with water *ad libitum*. Ethical committee approval was obtained from IAEC of C.L. Baid Metha College of pharmacy. The approval number is (IAEC/XLVI/08/CLBMCP/2015 dated: 20/08/2015)

## MATERIALS AND METHODS

**Preparation of Plant Extract**

The sprout of seeds of *Vigna radaita* were collected from local source, Tamil Nadu in December. The plant material was identified and authenticated by Dr.P. Jayaraman., Retd. Professor, Presidency College, Chennai–600005, Tamilnadu. [PARC/2014/2095]. The powdered sprouted seed were dried and extracted with water: ethanol (40:60) in Soxhlet's apparatus at room temperature for 24 hours. The extract was stored at 0-4°C. The yield of the hydro alcoholic extract was 6.84% w/w and it was preserved in refrigerator for further use.

**Preliminary Phytochemical Analysis of HAEVR**

The HAEVR was subjected to preliminary phytochemical screening for the presence or absence of phytoconstituents such as alkaloids, carbohydrates, flavonoids, gums and mucilage, tannins, phenols, saponin, terpenes, proteins, steroids, glycosides and sterols.

**Acute Oral Toxicity Studies**

The Acute Oral Toxicity Study was done according to the OECD guidelines 423. A single administration of 2000 mg/kg b.w /p.o of the HAEVR for three days in 3 female wistar albino rats and observed for 14 days.

Body weights of rats before and after administration were observed for morbidity and mortality. Any changes in skin, fur, eyes, mucous membrane, respiratory, circulatory, autonomic and rheumatism,
central nervous system, motor activity and behaviour pattern were observed and also signs of tremors, convulsion, salivation, diarrhoea, lethargy, sleep and coma were noted.

**Experimental Design:**

Adult male wistar albino rats weighed 180-220gms were divided into five groups of six animals each. The development of diabetes was confirmed after 72 hours of the Streptozotocin injection. Animals with more than 250 mg/dl was considered as diabetic (Kumar *et al*., 2008; Gupta *et al*., 2004) (Table 1).

**Table 1: Experimental Design**

| Groups | Treatment |
|---|---|
| I | Control animals treated with 1% w/v SCMC. |
| II | STZ (55 mg/kg b.w., i.p) induced diabetic animals. |
| III | STZ (55 mg/kg b.w., i.p) induced diabetic animals treated with Glibenclamide 5mg/kg b.w/p.o. |
| IV | STZ (55 mg/kg b.w., i.p) induced diabetic animals treated with HAEVR 200mg/kg b.w/ p.o. |
| V | STZ (55 mg/kg b.w., i.p) induced diabetic animals treated with HAEVR 400mg/kg b.w/ p.o. |

**Biochemical and Histopathology Analysis:**

On $28^{th}$ day animal was sacrificed by decapitation, blood was collected and serum was separated to study the biochemical parameters. The blood glucose level, serum Protein (Lowry *et al*., 1951), serum lipids (Folch *et al*., 1957) cholesterol (Zlatkis *et al*., 1953), triglyceride, HDL (Burstein *et al*., 1970), VLDL, LDL (Friedwald *et al*., 1972), SGOT, SGPT (Reitman *et al*., 1957), plasma Creatinine (Slot, 1965) and urea (Wybenga *et al*., 1971) were estimated. Histopathology studies of liver, kidney and pancreas were carried out by using standard procedure.

## RESULTS

**Phytochemical analysis:**

The result of preliminary phytochemical analysis of HAEVR shows presence of Alkaloids, phenols, flavanoids, steroids and absence of Carbohydrates, Steroids, Sterols, Phenols, Flavanoids, Glycosides, Terpenes, gums and mucilage. (Table 2).

**Oral Acute toxicity studies:**

There was no considerable change in body weight before and after treatment and no signs of toxicity was observed. (Table 3).

**Body weight:**

At initial day body weight in group I was compared with group II, III, IV and V were significantly decreased ($p<0.001$). The bodyweight in group II was compared with group III and IV were decreased ($p<0.001$). The bodyweight in group II was compared with group V ($p<0.001$) significantly increased. (Table 4) (Figure 1).

On 7$^{th}$ day body weight in group I was compared with group II, group III, IV and V ($p<0.001$) significantly decreased. The bodyweight in group II was compared with group IV (ns), III and V were significantly increased ($p<0.001$). (Table 4) (Figure 1).

On 14$^{th}$ day body weight in group I was compared with group II, III, IV and V were significantly decreased ($p<0.001$). The bodyweight in group II was compared with group IV (ns), III and V ($p<0.001$) were significantly increased. (Table 4)(Figure 1)

On 21$^{st}$ day body weight in group I was compared with group II, group III, group IV and group V were significantly decreased ($p<0.001$). The bodyweight in group II was compared with group IV (ns), III and V ($p<0.001$) were significantly increased. (Table 4)(Figure 1).

On 28$^{th}$ day weight in group I was compared with group II, III, IV and V ($p<0.001$) significantly decreased. The bodyweight in group II was compared with group IV (ns), III and V ($p<0.001$) were significantly increased. (Table 4)(Figure 1).

**Blood glucose level:**

At initial day blood glucose levels in group I was compared with group II, III, IV and V were significantly increased. The blood glucose levels in group II was compared with group III were increased**.** The blood glucose levels in group II was compared with group IV and V were decreased. (Table 5) (Figure 2).

On 7$^{th}$ day blood glucose levels in group I was compared with group II, III, IV and V ($p<0.001$), were significantly increased. The blood glucose levels in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased**.** (Table5) (Figure 2).

On 14$^{th}$ day blood glucose levels in group I was compared with group II, III, IV and group V ($p<0.001$), were significantly increased. The blood glucose levels in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table5) (Figure 2).

On 21$^{st}$ blood glucose levels in group I was compared with group II, III, IV and group V ($p<0.001$), were significantly increased. The blood glucose levels in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table5) (Figure 2).

On 28$^{th}$ blood glucose levels in group I was compared with group III (ns), II, IV and V ($p<0.001$), were significantly increased. The blood glucose levels in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table5) (Figure 2).

**Biochemical parameters:**

The serum cholesterol level in group I was compared with group II, III, IV and V are significantly increased ($p<0.001$). The serum cholesterol level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 6) (Figure 3). The serum HDL level in group I was compared with group III ($p<0.01$), II, IV and V ($p<0.001$) were significantly decreased. The HDL level in group II was compared with group III group IV and group V ($p<0.001$) were significantly increased. (Table 6) (Figure 4).

The serum LDL level in group I was compared with group III ($p<0.01$), II, IV and V ($p<0.001$) were significantly increased. The serum LDL level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 6) (Figure 5).

The serum VLDL level in group I was compared with group II, III, IV and V ($p<0.001$) were significantly increased. The serum VLDL level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 6) (figure 6).

The serum triglyceride level in group I was compared with group II, III, IV and V ($p<0.001$) were significantly increased. The serum triglyceride level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 6) (Figure 7).

The serum total protein level in group I was compared with group II ($p<0.01$), III, IV and V (ns) were increased. The serum total protein level in group II was compared with group IV ($p<0.001$), III and V ($p<0.01$) were significantly decreased. (Table 7) (Figure 8).

The serum creatinine level in group I was compared with group II, III, IV and V ($p<0.001$) were significantly increased. The serum creatinine level in group II was compared with group III, IV and

V ($p<0.001$) were significantly decreased. (Table 7) (Figure 9).

The serum urea level in group I was compared with group II, III, IV and V ($p<0.001$) were significantly increased. The serum urea level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 7) (Figure 10).

The SGOT level in group I was compared with group II, III, IV and V ($p<0.001$) were significantly increased. The SGOT level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 7)(Figure 11).

The SGPT level in group I was compared with group II, III, IV and V were significantly increased ($p<0.001$). The SGPT level in group II was compared with group III, IV and V ($p<0.001$) were significantly decreased. (Table 7) (Figure 12).

**Histopathology**

The histopathology of STZ induced pancreas of control animals showed normal islets and acini whereas diabetic control showed damages and atrophy islets with acini. Diabetic animal treated with glibenclamide (5mg/kg b.w/p.o) showed preserved normal islets in pancreas whereas HAEVR 200mg/kg/p.o treated animals showed small pancreatic islet and HAEVR 400mg/kg/p.o treated animals showed hyperplastic (Figure 13).

Histological evaluation of kidney sections of extract drenched animals revealed almost no morphological difference from control. The variation in mean nuclear diameter of kidney cells of treated animals was, also, not significantly different from control. Although the results of biochemical and histological results may indicate that drenching HAEVR extract is safe on rat kidney (Figure 14).

Histopathological examinations of diabetic animals liver showed centrilobular necrosis accompanied by fatty changes and ballooning degeneration in the remaining hepatocytes in the liver of rats treated with STZ (55mg/kg b.w/p.o) were much of intensity and which was recovered with the treatments HAEVR. (Figure 15).

**Table 2. Preliminary phytochemical analysis of HAEVR:**

| Sl.No | Phytochemical Tests | HAEVR |
|---|---|---|
| 1 | Test for Alkaloids, phenols, flavanoids, steroids | +ve |
| 2 | Test for Carbohydrates, Steroids, Sterols, Phenols, Flavanoids, Glycosides, Terpenes, gums and mucilage | -ve |

**Table 3. Acute oral toxicity study of HAEVR:**

| Si. No. | Treatment group | Dose | Weight of animal in gms | | Signs of toxicity | Onset of toxicity | Reversible or irreversible | Duration |
|---|---|---|---|---|---|---|---|---|
| | | | Before test | After test | | | | |
| 1. | HAEVR | 2g/kg | 180 | 190 | No signs of toxicity | Nil | Nil | 14 days |
| 2. | HAEVR | 2g/kg | 175 | 190 | No signs of toxicity | Nil | Nil | 14 days |
| 3. | HAEVR | 2g/kg | 190 | 200 | No signs of toxicity | Nil | Nil | 14 days |

**Table 4. Effect of HAEVR on body weight of STZ induced diabetic rats**

| Group | Body weight (gm.) | | | | |
|---|---|---|---|---|---|
| | Day – 0 | Day – 7 | Day – 14 | Day – 21 | Day – 28 |
| I | 186.2±1.939 | 188.2±2.272 | 196.5±0.7638 | 204.0±1.183 | 210.2±1.400 |
| II | 183.2±4.110 a*** | 152.5±1.432 a*** | 133.5±1.088 a*** | 124.7±0.8819 a*** | 121.3±1.542 a*** |
| III | 182.7±2.108a*** b*** | 172.2±2.023 a*** b*** | 173.5±1.232a*** b*** | 181.7±0.8819 a*** b*** | 191.0±1.880 a*** b*** |
| IV | 180.0±3.651a*** b*** | 160.7±2.246 a*** b ns | 162.8±0.9458a*** b*** | 168.2±0.7923 a*** b*** | 175.0±1.238 a*** b*** |
| V | 184.0±3.317a*** b*** | 166.3±1.994 a*** b*** | 169.8±0.9098a*** b*** | 175.8±1.400 a**** b*** | 181.2±1.447 a*** b*** |

Values are expressed as mean ± SEM of 6 animals. Comparisons were made between the following: a - Group I vs. II, III, IV and V, b - Group II vs. III, IV and V. Statistical Significance test for comparison was done by one way ANOVA followed by Dunnets 't' test. Where *p< 0.05, **p< 0.01, ***P<0.001, ns-non significant.

**Table 5. Effect of HEAVR on blood glucose level in STZ induced diabetic rats**

| Group | Blood glucose (mg/dl) | | | | |
|---|---|---|---|---|---|
| | Day – 0 | Day – 7 | Day – 14 | Day – 21 | Day – 28 |
| **I** | 91.00±3.120 | 83.50±1.668 | 95.83±3.646 | 91.33±2.996 | 95.50±3.828 |
| **II** | 100.3±5.649 | 250.8±1.922a*** | 285.2±2.613 a*** | 290.7±1.520 a*** | 297.8±1.701 a*** |
| **III** | 105.3±6.586 | 171.0±1.549a*** b*** | 142.0±1.461 a*** b*** | 121.3±1.783 a*** b*** | 103.3±1.856 a$^{ns}$ b*** |
| **IV** | 98.83±6.290 | 220.5±1.607 a*** b*** | 201.2±2.257 a*** b*** | 159.8±1.515 a*** b*** | 130.5±1.688 a*** b*** |
| **V** | 92.83±3.390 | 180.8±1.838a*** b*** | 161.0±1.528 a*** b*** | 120.0±1.065 a*** b*** | 109.8±2.272 a** b*** |

**Table 6. Effect of HAEVR on Total cholesterol, HDL, LDL, VLDL, Triglycerides.**

| Groups | Total cholesterol (mg/dl) | HDL (mg/dl) | LDL(mg/dl) | VLDL (mg/dl) | Triglycerides( mg/dl) |
|---|---|---|---|---|---|
| Group I | 108.3±0.8242 | 60.60±0.7169 | 82.02±0.5598 | 16.34±0.2994 | 86.18±0.3630 |
| Group II | 206.3±0.6184 a*** | 33.46±0.3918 a*** | 141.6±0.8738 a*** | 40.87±0.4726 a*** | 170.5±0.5911 a*** |
| Group III | 119.3±0.4665 a*** b*** | 57.68±0.3218 a** b*** | 85.08±0.2794 a** b*** | 19.48±0.3522 a*** b*** | 90.37±0.4061 a*** b*** |
| Group IV | 159.3±0.9684 a*** b *** | 42.56±0.2840 a*** b*** | 111.0±0.4890 a*** b*** | 29.54±0.2956 a*** b*** | 123.6±0.4776 a*** b*** |
| Group V | 136.8±0.7151 a*** b*** | 54.88±0.5408 a*** b *** | 91.93±0.4630 a*** b*** | 21.46±0.4489 a*** b*** | 96.34±0.4439 a*** b*** |

**Table 7. Effect of HAEVR on Total protein, Creatinine, Urea, SGOT, SGPT.**

| Groups | Total protein (mg/dl) | Creatinine (mg/dl) | Urea(mg/dl) | SGOT (U/L) | SGPT (U/L) |
|---|---|---|---|---|---|
| Group I | 6.405±0.1967 | 0.4983±0.02822 | 19.08±0.2544 | 68.10±0.5008 | 41.44±0.5317 |
| Group II | 5.690±0.1119 a** | 1.822±0.02774 a*** | 52.60±0.4275 a*** | 139.8±0.6129a*** | 98.96±0.4503 a*** |
| Group III | 6.635±0.1179 a$^{ns}$ b*** | 0.6667±0.01856 a***b*** | 23.29±0.2846 a*** b*** | 97.86±0.4566 a*** b*** | 60.86±0.6440 a*** b*** |
| Group IV | 6.442±0.1196 a$^{ns}$ b** | 1.272±0.02960 a*** b*** | 43.84±0.5212 a*** b*** | 89.37±0.4797 a*** b*** | 52.55±0.4378 a*** b*** |
| Group V | 6.512±0.1104 a$^{ns}$ b** | 0.8817±0.01701 a***b*** | 27.28±0.5582a*** b*** | 82.49±0.5245 a*** b*** | 48.75±0.4090 a*** b*** |

**Figure 1. Effect of HAEVR on body weight of STZ induced diabetic rats**

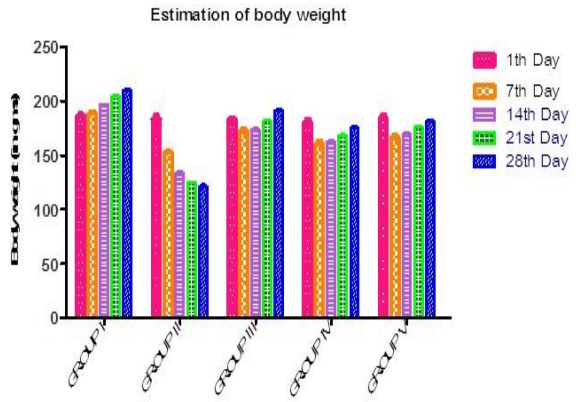



**Figure 2. Effect of HEVR on blood glucose level in STZ induced diabetic rats**

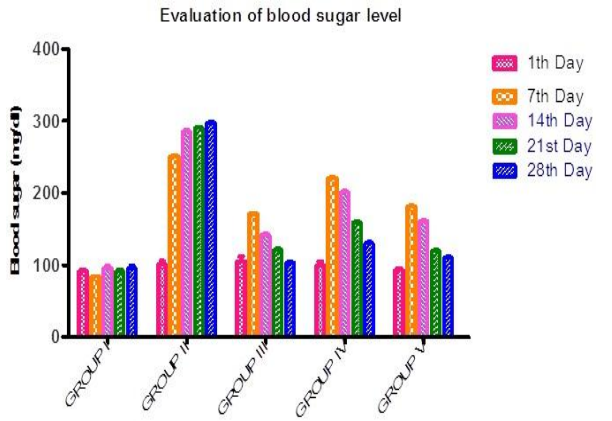


**Figure 3.**

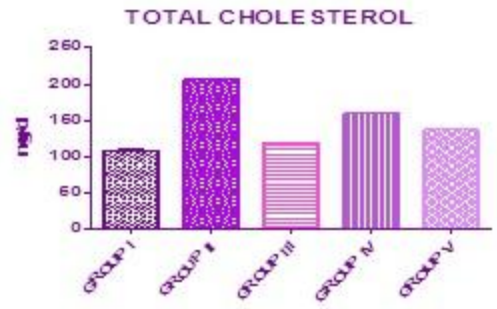



**Figure 4.**

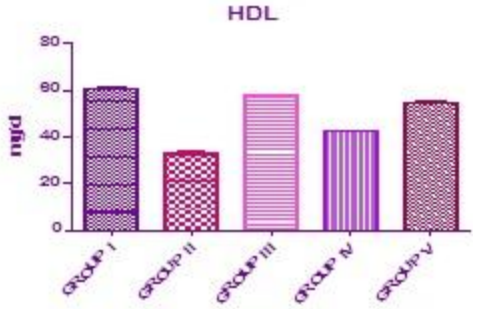



**Figure 5.**

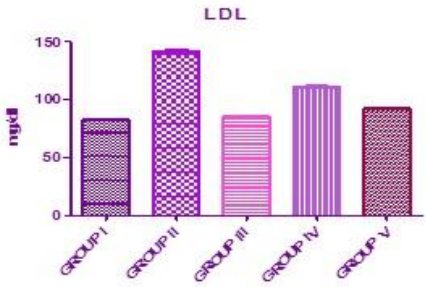



**Figure 6.**

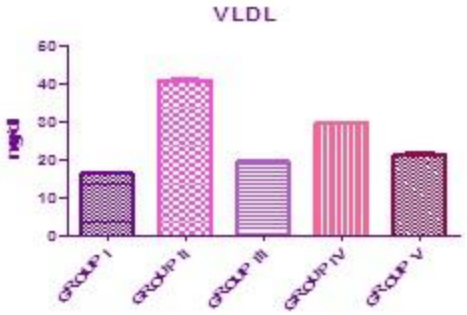



**Figure 7.**


**Figure 8.**

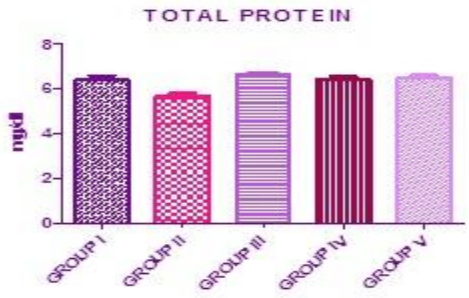



**Figure 9.**

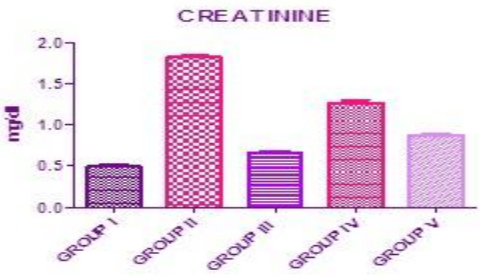



**Figure 10.**


**Figure 11.**

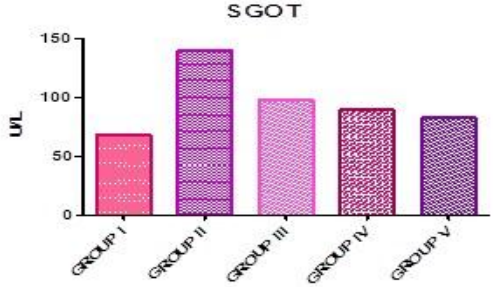



**Figure 12.**

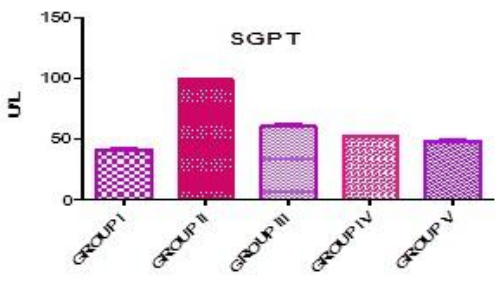


**Figure 13. Histopathology of Pancreas**

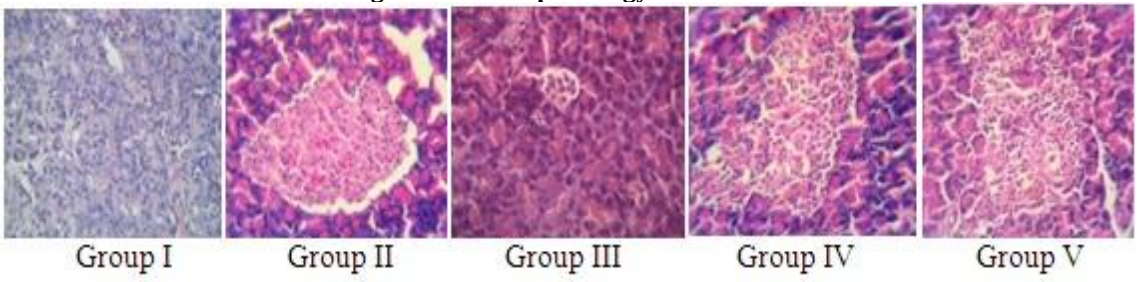



**Figure 14. Histopathology of Kidney**

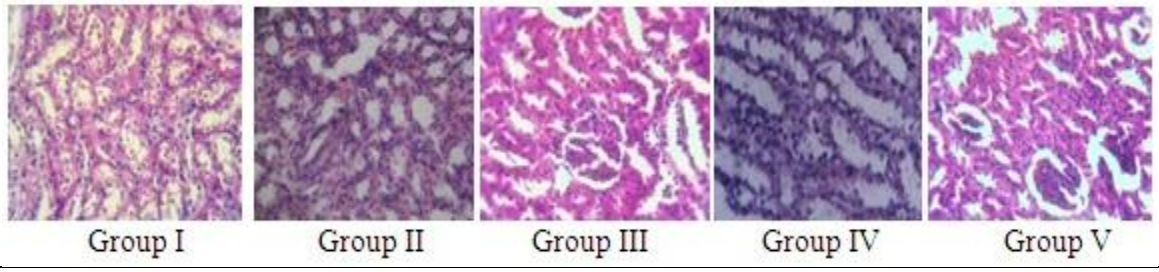
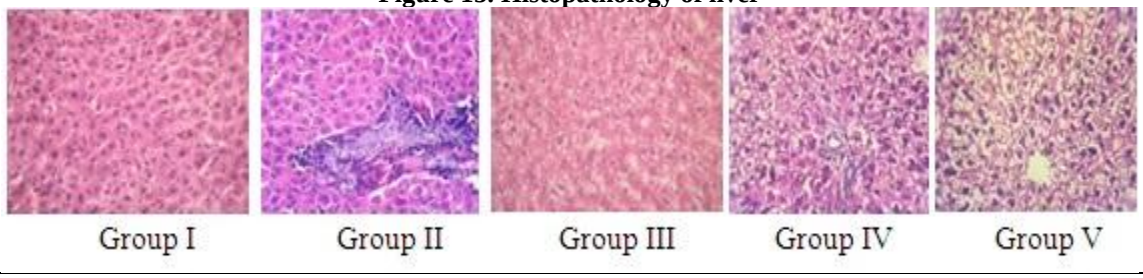



**Figure 15. Histopathology of liver**




## DISCUSSION

Diabetes is the condition in which the body does not properly process food for use energy. The term diabetes mellitus describes a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion insulin action, are both the chronic hyperglycemia of diabetes is associated with relatively specific long term micro vascular.

The increase in number of diabetic patients has motivated to find new methods to cure diabetes. Inspite of the presence of known anti-diabetic medicine in the pharmaceutical market, remedies from medicinal plants are used with success to treat this disease (Bhattaram *et al*., 2002). This attribute antihyperglycemic effects of these plants is due to their ability to restore the function of pancreatic tissues by causing an increase in insulin output or inhibit the intestinal absorption of glucose or to the facilitation of metabolites in insulin dependent processes. Hence, treatment with herbal drugs has an effect on protecting beta cells and smoothing out fluctuation in glucose levels (Elder, 2004). The present study involved the evalulation of anti-diabetic activity of sprout of *Vigna radaita* in STZ induced diabetic rats.

Preliminary Phytochemical analysis showed the presence of flavonoids, which act as insulin secretagogues or insulin mimetics, probably by influencing the pleiotropic mechanisms to attenuate diabetic complications. Flavonoids may be responsible for the stimulation of glucose uptake in peripheral tissues and regulation of the activity and/or expression of the rate-limiting enzymes involved in carbohydrate metabolism (Naik *et al*., 2003). Acute oral toxicity study of HAEVR did not exhibit mortality or any profound toxic reactions at a dose of 2000mg/kg/p.o.

The mechanism by which STZ brings about its diabetic state include selective destruction of pancreatic insulin secreting beta cells, which make cells less active and lead to poor glucose utilization by tissues (Marles RJ & Farnsworth NR, 1995). STZ induced diabetic model resembles type I diabetes with final symptoms of insulin deficiency. Long term treatment (28 days) with active fraction of HAEVR resulted in mild improvement in plasma insulin levels. This suggests that *Vigna radaita* like glibenclamide stimulates insulin secretion from the

remnant beta cells of islets of Langerhans (Shanmugasundaram *et al*., 1990) or the drug may be mimicking one or more actions of insulin at the insulin receptor level or/and it may be influencing one or more post receptor events.

Experimental induction of hyperglycemia with STZ is associated with the characteristic loss of body weight which is due to loss or degradation of structural proteins it leads to increased muscle wasting and due to loss of tissue protein, as the structural proteins are known to contribute to body weight. Diabetic rats treated with glibenclamide and HAEVR showed increased body weight when compared to untreated diabetic animals. It may be due to increased insulin secretion and glycemic control of HAEVR.

The mechanism involved in suppressing blood glucose levels may be by the following possibilities. Reduced glucose transport or absorption from the gut, extra pancreatic action probably by stimulation of glucose utilization in peripheral tissues, increase in glycogenic or glycolytic enzyme activities in peripheral tissues, decrease in the secretion of counter-regulatory hormones like glucagon, growth hormones. The glibenclamide, stimulating insulin secretion from pancreatic β cells principally by inhibiting ATP sensitive $K_{ATP}$ channels in the plasma membrane and decreases the blood glucose level (Shanmugasundaram *et al*., 1990). Blood glucose level decreased significantly in glibenclamide and HAEVR treated diabetic rats and the histopathology of pancreas showed normal islets in pancreas with normal anatomy compared with normal rats which may be due to the anti-diabetic activity.

In diabetes, hyperglycaemia is accompanied with dyslipidemia (Yamamoto *et al*., 1981) under normal circumstances; insulin activates the enzyme lipoprotein lipase, which hydrolyses triglycerides. However, in diabetic state lipoprotein lipase is not activated due to insulin deficiency, resulting in hyper triglyceridemia, and insulin deficiency is also associated with hyper cholesterolemia due to metabolic abnormalities. The dyslipidemia is characterized by increase in TC, LDL, VLDL, TG and fall in HDL which is observed in STZ induced diabetic rats (Padmini *et al*., 1982). The diabetic rats treated with glibenclamide and HAEVR showed reduced severity of dyslipidemia with decrease in TC, LDL, VLDL, TG and increase in HDL level (Kavalali *et al*., 2000; Bierman *et al*., 1996).

Both SGOT and SGPT enzyme levels get elevated during liver damage which is more in diabetic rats (Ellils *et al*., 1978). The diabetic rats treated with glibenclamide and HAEVR reduced the SGOT and SGPT level. The liver histopathology of STZ induced diabetic rats showed centrilobular necrosis accompanied by fatty changes and ballooning degeneration in the heptocytes which was reversed in diabetic rats treated with glibenclamide and HAEVR which indicates that the liver damage is reduced in HAEVR treated group.

The diabetic hyperglycemia induces elevation of the serum levels of urea and creatinine which are significant markers of renal dysfunction and reflecting a decline in the glomerular filteration rate (Muhammad *et al*., 2008) which is a complication in type II diabetes. The urea and creatinine level in glibenclamide treated animals showed decreased serum urea and creatinine level. The kidney histopathology sections revealed almost no morphological difference in mean nuclear diameter of kidney cells from control it indicates that drenching HAEVR extract is safe on rat kidney. Hence it is an evident that HAEVR reduced the complication of diabetes.

## CONCLUSION

The sprout of *Vigna radaita* reduces the blood glucose level and lipid profile like TC, TG, LDL, and VLDL, increase HDL. The *Vigna radaita* reduces SGOT, SGPT, total protein, creatinine and urea in STZ induced diabetic rats. Histopathology shows that the sprout of reduced liver and kidney damage which is common in diabetes.

Thus, it may be concluded that *Vigna radaita* produces significant anti-diabetic activity in Streptozotocin induced diabetic rats, which is comparable with that of Glibenclamide. Further work is necessary to elucidate the mechanism of action involved in the anti-diabetic activity of *Vigna radaita* with special references to phytochemical constituents.

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**Cite this article:**
Amudha P, Chinnadurai. Evaluation of anti-diabetic activity of hydro alcoholic extract of sprouts of seeds of *Vigna radiata* on streptozocin induced diabetes in wistar rats. *International Journal of Phytopharmacology*, 8(2), 2017, 71-79.
DOI: **http://dx.doi.org/10.21276/ijp.2017.8.2.4**