1. Introduction

1.1 Diabetes mellitus

Diabetes mellitus is a group of metabolic disorders characterized by elevated plasma glucose levels. Diabetes mellitus is prevalent in all countries of the world. More than 30 million people are said to be affected throughout the world with this disease. Diabetes mellitus is presently the most common, non communicable disease worldwide and is the fourth or fifth leading cause of death in most developed countries. The worldwide prevalence of diabetes mellitus has risen dramatically over the past two decades. According to the World Health Organization (WHO), more than 180 million people worldwide are currently living with this disorder, with these numbers looking set to double by the year 2030\textsuperscript{1}. In 2005, an estimated 1.1 million people died from diabetes with a projected rise in deaths of 50\% over the next 10 years. It is estimated that at least 1 in 20 deaths, globally and across all ages, are attributable to diabetes\textsuperscript{2}. About 80\% of all diabetes deaths occur in the low and middle income segments, with the greatest increases expected in Africa and Asia\textsuperscript{3,4}. The increase in incidence of diabetes in developing countries follows the trend of urbanization and lifestyle changes, perhaps most importantly a “Western-style” diet.

Despite of the large changes in the input and utilization of glucose, the blood glucose levels are maintained at constant level in response to hormonal signals. So, the concentration of glucose in the blood is subjected to tight regulation and homeostasis of glucose in blood is mainly maintained by cooperative activity of liver, muscle, kidney and adipose tissue and by the endocrine glands at the cellular and enzymatic levels.

The cells of our body use glucose for energy and are essential to normal function of the body. Nerve tissues are especially dependent on glucose as their source of energy and it is the principal fuel for retina of the eyes and the brain, which run almost entirely on
glucose. In fact, the brain cannot use protein or fat directly for its energy. Other cells are able to use fats or amino acids (proteins) if necessary. The brain responds to low blood sugar levels by calling for more glucose via the nervous system and pituitary gland stimulates the adrenal glands to release adrenaline (epinephrine). This in turn, stimulates the liver to adjust the glucose level in the blood. The glucose levels are determined by how fast glucose enters and leave the bloodstream. Glucose is absorbed from the intestines into the blood of the portal vein. As it passes through the liver, excess glucose is converted into glycogen. Glucose is stored in the body in the form of glycogen for future conversion into sugar and for subsequent use in muscular work or for liberating energy. Glycogen can be easily mobilized for metabolic processes and it is converted into glucose when needed by the tissues.

The endocrine function of the pancreas is to control the amount of sugar in the blood by producing insulin (pancreas also produces another hormone) and secreting it into the bloodstream. The concentration of glucose in the blood is maintained through the action of Insulin produced by specific cells in the pancreas, called the Islets of Langerhans. Without insulin, the glucose level rises in the bloodstream, but the body is unable to use it for energy production. Insulin enables the body to transport glucose inside cells where it can be used for energy metabolism. If the sugar level is too high or rises too fast, insulin is produced into the bloodstream. Insulin promotes glucose utilization and protein synthesis, along with other mechanisms. As the blood glucose level rises after a meal, insulin is released by the pancreas to lower the glucose level. As the blood glucose falls, the insulin released from the pancreas decreases. Overproduction of insulin by the pancreas burns off too much sugar (reduces the blood sugar below normal) in the blood and leave the individual with a malfunction of sugar metabolism and an array of unpleasant symptoms. If the pancreas is not functioning properly (inadequate secretion of insulin) the result is improper metabolism of carbohydrates and fats, which can eventually lead to diabetes.
The liver plays glucostatic role in response to hormonal signals and by levels of glucose itself, while the kidney operates by way of filtration and reabsorption. Glucose metabolism is defective in two very common metabolic diseases that is, obesity and diabetes, which in turn contribute to factors in the development of number of major medical complications including atherosclerosis, hypertension, small vessel diseases, kidney diseases, blindness etc.

Consumption of diets highly enriched with saturated fats or simple sugars (e.g. glucose) can increase insulin concentrations, enhance adipose tissue deposition, reduce insulin sensitivity and impair glucose tolerance. These effects are seen even if total energy intake is not increased, but are more pronounced if this is greater. High – fat feeding can also enhance diabetic features in rodents treated neonatal with streptozotocin or with ventromedial hypothalamic lesions5,6.

Diabetes mellitus is a disease characterized by disorder metabolism and abnormally high blood sugar levels resulting from body’s inability to produce or properly use insulin7. As the number of people with diabetes multiplies worldwide, the disease takes an ever increasing proportion of national and international health care budget. It is becoming the third “killer” of mankind after cancer and cardiovascular diseases, because of its high prevalence, morbidity and mortality8. Though insulin is widely accepted as an ideal choice for treatment of diabetes mellitus, the difficulty of repeated administration led to the search for the new hypoglycemic agents.

Longstanding diabetes is associated with alterations in mitochondrial metabolism that result in both increased formation of reactive oxygen species (ROS) and failure of bioenergetics. In particular, diabetes causes dysfunction of mitochondria in those tissues which are highly dependent on aerobic metabolism such as heart, brain and skeletal muscle. Complications from diabetes mellitus such as cardiovascular
disease, peripheral vascular disease, stroke, diabetic neuropathy, amputations, renal failure and blindness are on the increase. About 75% of deaths among men with diabetes and 57% among women with diabetes are attributed from cardiovascular diseases (CVD). Therefore, it is certain that diabetes mellitus (DM) will be one of the most challenging health problems in the new millennium. Prevention and control programs are needed to stem the rising epidemic of DM and its complications.

In recent years, researchers turned attention specifically to oxidative stress and the key role it plays, as a common element in the pathogenesis of diabetes complications. Hyperglycemia generates reactive oxygen species which, in turn, causes membrane lipid peroxidation and degradation. Many of the complications of diabetes, including vascular atherosclerosis are closely related to oxidative stress and thus, antioxidants play an important role in the treatment of diabetes. Depending on the etiology of diabetes, factors leading to hyperglycemia include decreased insulin secretion, decreased glucose use and increased production of glucose. The metabolic deregulations associated with DM causes secondary pathophysiologic changes in multiple organ system that imposes a tremendous burden on the individual with diabetes and on health system. DM is the leading cause of end-stage renal disease, nontraumatic lower extremity amputation and adult blind.

1.2 Forms of Diabetes mellitus

Diabetes occurs in two main forms; type I (insulin deficiency) and type II (insulin resistance).

(a) Type 1 diabetes

Type 1 diabetes was previously termed insulin-dependent diabetes mellitus (IDDM). It develops when the body’s immune system destroys pancreatic β-cells, the only cells in the body that produce the hormone insulin to regulate blood glucose. This
form of diabetes usually strikes children and young adults, although disease onset can occur at any age. In adults, type 1 diabetes accounts for 5% to 10% of all diagnosed cases of diabetes. Risk factors for type 1 diabetes may be autoimmune, genetic, or environmental. The principal treatment of type 1 diabetes mellitus, even in its earliest stages, is the delivery of artificial insulin via injection combined with careful monitoring of blood glucose levels using blood testing monitors. Without insulin, diabetic ketoacidosis often develops which may result in coma or death. Treatment emphasis is now also placed on lifestyle adjustments such as diet and exercise which can hinder the progress of the disease, but not reverse it. Apart from the common subcutaneous injections, it is also possible to deliver the insulin by a pump, which allows continuous infusion of insulin 24 hours a day at preset levels and the ability to program doses of insulin as needed at meal times. An inhaled form of insulin was approved by the food and drug administration (FDA) in January 2006, although it was discontinued for business reasons in October 2007. Non-insulin treatments, such as monoclonal antibodies and stem-cell based therapies, are effective in animal models but have not yet completed clinical trials in humans.

(b) Type 2 diabetes

This type of diabetes was formerly called non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes mellitus. It results from the body’s ineffective use of insulin. Type 2 diabetes mellitus is mainly due to insulin resistance or reduced insulin sensitivity, combined with relatively reduced insulin secretion which in some cases becomes obsolete. The defective responsiveness of the body tissues to insulin almost certainly involves the insulin receptor in the cell membranes. However, the specific defects are not known but the low receptor density is due to destruction by
ketonic bodies\textsuperscript{11}. In the early stages of type 2 diabetes, the predominant abnormality is reduced insulin sensitivity, characterized by elevated levels of insulin in the blood. At this stage hyperglycemia can be reversed by a variety of measures and medication that improve insulin sensitivity or reduce glucose production by the liver\textsuperscript{11}. As the disease progresses, the impairment of insulin secretion worsens and therapeutic replacement of insulin often becomes necessary. Type 2 diabetes mellitus comprises 90\% of people with diabetes around the world, and is largely as a result of excess body weight and physical inactivity\textsuperscript{11}.

Type I requires insulin injection, whereas type II diabetes can often be controlled using oral drug alone.

1.3 Current therapies

1.3.1 Injectable antidiabetic agents

a. Insulin

Insulin was discovered in 1921 by Banting and Best. It is synthesized in the \( \beta \) cell of the pancreatic islets as a single chain peptide called pre pro insulin with 110 amino acids from which 24 amino acids are first removed to produce pro insulin\textsuperscript{12}. Insulin is a two chain polypeptide having 51 amino acids with molecular weight of about 6000. The A chain has 21 amino acids while the B chain has 30 amino acids The connecting peptide or the C chain having 35 amino acids is split of by proteolysis and secreted in to the blood\textsuperscript{13}.

The C-chain is removed enzymatically resulting in two strands joined by intermolecular disulfide bonds, giving active insulin. Insulin is utterly necessary for controlling type I diabetes and it is usually derived from pork or bovine sources. Insulin is often administered as a complex with protamine and zinc. This slows down its action, providing longer glucose control\textsuperscript{11}. 
b. Amylinagonists (Pramlintide)

Pramlintide is a synthetic analog of the β-cell hormone amylin. It is administered subcutaneously before meals, inhibits glucagons production in a glucose-dependent fashion and predominantly decreases postprandial glucose excursions.¹⁴

1.3.2 Oral anti diabetic agents

Classes of oral antidiabetic agents include sulfonylureas, biguanides, α-glucosidase inhibitors, glinides and thiazolidinediones.

a. Sulfonylureas

It was observed that certain sulphonamides (antibacterial) often resulted in hypoglycemia and hence non-antibacterial sulfonylureas which also reduced blood glucose levels were developed.¹¹ These drugs function by stimulating the release of endogenous reserves of insulin from the pancreas and therefore are only effective in patients who are still able to synthesize and secrete insulin. This is achieved by binding to specific receptors that are associated with the potassium channel in the β cell membrane. Binding of sulphonylurea inhibits the efflux of potassium ion through the channel and result in depolarization. Depolarization inturn opens a voltage gated calcium channel and results in calcium influx and release of insulin.¹⁵

Unfortunately, sulfonylureas do not always succeed in controlling diabetes. Common side effects include; hypoglycemia, increased appetite and weight gain, as well as an increased cardiovascular risk.¹⁶ Tolbutamide is an example for sulfonylurea.

b. Biguanides

Similarly to sulfonylureas, the discovery of the biguanides was also serendipitous. It was found that a guanidine derivative lowered blood glucose levels in rabbits. This led to the development of phenformin. Unlike sulfonylureas, biguanides lower blood glucose levels without stimulating the release of insulin from the pancreas.
Metformin and phenformin were introduced for the treatment of diabetes in the 1950s\textsuperscript{11}. Phenformin, however, was withdrawn from clinical use shortly after introduction, leaving metformin as the only ‘surviving’ example in this class. Metformin reduces blood glucose concentrations by increasing glucose uptake in the peripheral muscles and decreasing the amount of glucose produced and released in the liver i.e. suppress hepatic gluconeogenesis\textsuperscript{17}.

Metformin showed the added benefit of reducing weight gain and even caused weight loss in certain patients. Gastrointestinal side effects such as a metallic taste, nausea, abdominal pain and diarrhoea occur in varying severity in up to 30\% of patients\textsuperscript{16}.

\begin{center}
\begin{tikzpicture}
\node (phenformin) at (0,0) {\includegraphics[width=0.4\textwidth]{phenformin.png}};
\node (metformin) at (1.5,0) {\includegraphics[width=0.4\textwidth]{metformin.png}};
\end{tikzpicture}
\end{center}

\textbf{c. $\alpha$-Glucosidase Inhibitors}

The $\alpha$-glucosidase inhibitors such as acarbose and miglitol inhibit the action of intestinal enzymes that break down carbohydrates. These oral antidiabetic agents delay glucose absorption and are particularly useful for patients with postprandial hyperglycemia (high blood sugar levels after eating). However, they are not as effective as sulfonylureas and biguanides in providing long-term control of blood glucose levels\textsuperscript{13}.

d. Glinides

Like the sulfonylureas, the glinides stimulate insulin secretion, although they bind to a different site within the sulfonylurea receptor. They have a shorter circulating half-life than the sulfonylureas and must be administered more frequently\textsuperscript{18}.
e. Thiazolidinediones

Thiazolidinediones (TZDs), also known as glitazones, bind to a nuclear receptor known as peroxisome-proliferator-activated receptor gamma. Thiazolidinediones are insulin sensitizers that increase insulin sensitivity and action in liver, muscle and fatty tissues to endogenous and exogenous insulin\(^{19}\). This has several downstream effects including promoting insulin-stimulated glucose uptake by skeletal muscle cells. Thus, these compounds decrease insulin resistance. Furthermore, TZDs also slightly reduce blood pressure, enhance fibrinolysis and improve endothelial function\(^{20-22}\). Some adverse effects of TZDs include; weight gain, edema and anemia\(^{23}\). This class of drugs is currently represented by rosiglitazone and pioglitazone.

![Chemical structures of rosiglitazone and pioglitazone](image)

1.3.3 Traditional Medicines in the management of diabetes

Medicinal plants have been used virtually in all cultures as a source of medicine\(^{24}\). It has become increasingly evident in recent years that a full spectrum of therapeutic agents for the treatment and prevention of human disease is far from being complete. In an attempt to fill in the gap, drug development research has now focused on traditional herbal remedies as a potential source for new and more effective medical therapies. Medicinal plants are the most exclusive source of life saving drugs for the majority of the world's population India being rich in its plant wealth. In developing
countries, 80% populations are using traditional medicine in primary medical problems\textsuperscript{25-26}. Researchers always aim to bring out cost-effective and efficacious medicines for the benefit of mankind. Pure and isolated plant constituents have given various useful drugs and are of great importance.

Plant is considered as a biosynthetic laboratory, as divergent chemical entities are synthesized in them. The natural plant products often serve as chemical models or templates for the design and total synthesis of new drug entities. Crude drugs are usually subjected to a suitable method of extraction and purification for the isolation of phytopharmaceuticals, which should be incorporated as active ingredients in the modern system of medicine.

Medicinal plants have curative properties due to the presence of various complex chemical substances of different composition, which are found as secondary plant metabolites in one or more parts of these plants. These plant metabolites, according to their composition, are grouped as alkaloids, glycosides, steroids, essential oils, etc. Among these alkaloids form the largest group.

According to the WHO, more than 70% of the world’s populations rely on traditional medicine to satisfy their principal health needs. Despite considerable progress in the management of diabetes mellitus by synthetic drugs, the search for indigenous natural anti-diabetic agents is still going on. In addition to oral agents and insulin therapy, phytotherapy is an alternative that offers a wide range of natural resources with hypoglycemic effects; some plants provide materials recommended for people with diabetes. The use of medicinal plants for treatment of diabetes mellitus dates back from the Ebres papyrus about 1550 B.C\textsuperscript{27}. In the discovery and development of new drugs for the treatment of many diseases, medicinal plants have been considered the leading sources because of their chemical and pharmacological
diversity\textsuperscript{28}. Even at present the use of medicinal plants for treatment of diabetes is common because, plant medicines are frequently considered to be less toxic and more free from side effects as well as low cost than their synthetic counterparts\textsuperscript{29,30}.

Herbal remedies are beneficial to patients with type 2 diabetes and, in addition to the diet prescribed by a doctor they can help glucose homeostasis, but they cannot replace insulin and oral medication. A great number of medicinal plants used in the control of diabetes mellitus have been reported\textsuperscript{31}. Several plants have been identified as the potential source of drugs in Indian system of Ayurveda medicine for the treatment of diabetes. The plants provide a potential source of hypoglycemic drugs because many plants and plant derived compounds have been used in the treatment of diabetes. Hypoglycemic natural products comprise flavonoids, xanthones, triterpenoids, alkaloids, glycosides, alkyl disulfides, aminobutyric acid derivatives, polysaccharides and peptides. Many conventional drugs of today, such as atropine, ephedrine, etc., have been derived from proteolytic molecules in medicinal plants. The discovery of the widely used hypoglycemic drug, metformin came from the traditional approach through the use of \textit{Galega officinalis}\textsuperscript{32}.

Worldwide over 1200 species of plants have been recorded as traditional medicine for diabetes\textsuperscript{33}. Some of these plants have been evaluated in laboratories and in a number of cases their efficacy has been confirmed, for instance \textit{Moringa stenopetala}\textsuperscript{34}, \textit{Psacalium decompositum}\textsuperscript{35}, \textit{Syzigium alternifolium}\textsuperscript{36}, \textit{Panax ginseng}, \textit{Opuntica cactus}, \textit{Tecoma stans}, \textit{Syzigium cumini}\textsuperscript{37}. Specific chemical constituents of these plants, such as polysaccharides, alkaloids triterpinoids, saponins, flavonoids and xanthones are believed to be responsible for the hypoglycemc effect\textsuperscript{37}.

Extracts of various plants have been shown to produce hypoglycemia in normal and experimental diabetic animals. Some of the commonly studied plants are
Momordica charantia, Allicin cepa, Allium sativum, Ficus begalensis, Eugenia jambolana, Abroma augusta, Azadirachta indica, Coccinia indica, Curcuma longa indica and Ocimum sanctum. Traditional plant medicines are used throughout the world for a range of diabetic presentations. Many Indian plants have been investigated for their beneficial use in different types of diabetes and reported in numerous scientific journals.

A list of medicinal plants with antidiabetic and related beneficial effects is given in table 1.

Table 1

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Plant Name</th>
<th>Antidiabetic and other beneficial effects in traditional medicine</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>01.</td>
<td><em>Annona squamosa</em></td>
<td>Hypoglycemic and anti-hyperglycemic activities of ethanol leaf-extract, Increased plasma insulin level</td>
<td>39-41</td>
</tr>
<tr>
<td>02.</td>
<td><em>Artemisia pallens</em></td>
<td>Hypoglycemic, increases peripheral glucose utilization or inhibits glucose reabsorption</td>
<td>42</td>
</tr>
<tr>
<td>03.</td>
<td><em>Areca catechu</em></td>
<td>Hypoglycemic</td>
<td>43</td>
</tr>
<tr>
<td>04.</td>
<td><em>Beta vulgaris</em></td>
<td>Increases glucose tolerance in OGTT</td>
<td>44</td>
</tr>
<tr>
<td>05.</td>
<td><em>Boerhavia diffusa</em></td>
<td>Increase in hexokinase activity, decrease in glucose-6-phosphatase and fructose bis-phosphatase activity, increase plasma insulin level, antioxidant</td>
<td>45-47</td>
</tr>
<tr>
<td>06.</td>
<td><em>Bombax ceiba</em></td>
<td>Hypoglycemic</td>
<td>48</td>
</tr>
<tr>
<td>07.</td>
<td><em>Butea monosperma</em></td>
<td>Antihyperglycemic</td>
<td>49</td>
</tr>
<tr>
<td>08.</td>
<td><em>Camellia sinensis</em></td>
<td>Anti-hyperglycemic activity, antioxidant</td>
<td>50,51</td>
</tr>
<tr>
<td>09.</td>
<td><em>Capparis decidua</em></td>
<td>Hypoglycemic, antioxidant, hypolipidaemic</td>
<td>52</td>
</tr>
<tr>
<td>10.</td>
<td><em>Caesalpinia bonducella</em></td>
<td>Hypoglycemic, insulin secretagogue, hypolipidemic</td>
<td>53-55</td>
</tr>
<tr>
<td>11.</td>
<td><em>Coccinia indica</em></td>
<td>Hypoglycemic</td>
<td>56</td>
</tr>
<tr>
<td>12.</td>
<td><em>Embla officinalis</em></td>
<td>Decreases lipid peroxidation, antioxidant, hypoglycemic</td>
<td>57-59</td>
</tr>
<tr>
<td></td>
<td>Plant Name</td>
<td>Function</td>
<td>Page</td>
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</tr>
<tr>
<td>13.</td>
<td><em>Eugenia uniflora</em></td>
<td>Hypoglycemic, inhibits lipase activity</td>
<td>60</td>
</tr>
<tr>
<td>14.</td>
<td><em>Ficus bengalenesis</em></td>
<td>Hypoglycemic, antioxidant</td>
<td>61</td>
</tr>
<tr>
<td>15.</td>
<td><em>Gymnema sylvestre</em></td>
<td>Anti-hyperglycemic effect, hypolipidemic</td>
<td>62,63</td>
</tr>
<tr>
<td>16.</td>
<td><em>Hemidesmus indicus</em></td>
<td>Anti-snake venom activity, anti-inflammatory</td>
<td>64</td>
</tr>
<tr>
<td>17.</td>
<td><em>Hibiscus rosasinensis</em></td>
<td>Initiates insulin release from pancreatic beta cells</td>
<td>65</td>
</tr>
<tr>
<td>18.</td>
<td><em>Ipomoea batatas</em></td>
<td>Reduces insulin resistance</td>
<td>66</td>
</tr>
<tr>
<td>19.</td>
<td><em>Momordica cymbalaria</em></td>
<td>Hypoglycemic, hypolipidemic</td>
<td>67,68</td>
</tr>
<tr>
<td>20.</td>
<td><em>Murraya koenigii</em></td>
<td>Hypoglycemic, increases glycogenesis and decreases gluconeogenesis and</td>
<td>69</td>
</tr>
<tr>
<td></td>
<td></td>
<td>glycogenolysis</td>
<td></td>
</tr>
<tr>
<td>21.</td>
<td><em>Phaseolus vulgaris</em></td>
<td>Hypoglycemic, hypolipidemic, inhibit alpha amylase activity, antioxidant</td>
<td>70-72</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Altered level of insulin receptor and GLUT-4 mRNA in skeletal muscle</td>
<td></td>
</tr>
<tr>
<td>22.</td>
<td><em>Punica granatum</em></td>
<td>Antioxidant, anti-hyperglycemic effect</td>
<td>73</td>
</tr>
<tr>
<td>23.</td>
<td><em>Salacia reticulata</em></td>
<td>Inhibitotary activity against sucrase, α-glucosidase inhibitor</td>
<td>74</td>
</tr>
<tr>
<td>24.</td>
<td><em>Syzygium alternifolium</em></td>
<td>Hypoglycemic and antihyperglycemic</td>
<td>75</td>
</tr>
<tr>
<td>25.</td>
<td><em>Terminalia belerica</em></td>
<td>Antibacterial, hypoglycemic</td>
<td>76</td>
</tr>
<tr>
<td>26.</td>
<td><em>Terminalia chebula</em></td>
<td>Antibacterial, hypoglycemic</td>
<td>77</td>
</tr>
<tr>
<td>27.</td>
<td><em>Tinospora crispa</em></td>
<td>Anti-hyperglycemic, stimulates insulin release from islets</td>
<td>78</td>
</tr>
<tr>
<td>28.</td>
<td><em>Vinca rosea</em></td>
<td>Anti-hyperglycemic</td>
<td>79</td>
</tr>
<tr>
<td>29.</td>
<td><em>Withania somnifera</em></td>
<td>Hypoglycemic, diuretic and hypcholesterolemial</td>
<td>80</td>
</tr>
</tbody>
</table>
Structure of the active constituent having antidiabetic potential given in the table 2.

Table 2

<table>
<thead>
<tr>
<th>Plant name</th>
<th>Structure of the active constituents having antidiabetic potential</th>
<th>Active constituent</th>
<th>Potential beneficial effects</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Allium cepa</em></td>
<td></td>
<td>S-methyl cystein sulfoxide</td>
<td>Hypoglycemic, hyperlipidemic and antioxidant activity</td>
<td>81, 82</td>
</tr>
<tr>
<td></td>
<td><img src="image" alt="Structure of S-methyl cystein sulfoxide" /></td>
<td>Diphenylamine</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Allium sativum</em></td>
<td></td>
<td>S-allyl cystein</td>
<td>Antihypoglycemic and antioxidant activity</td>
<td>83</td>
</tr>
<tr>
<td></td>
<td><img src="image" alt="Structure of S-allyl cystein" /></td>
<td>Allicin (diallyl thiosulfimates)</td>
<td>Antihypoglycemic activity</td>
<td>84, 85</td>
</tr>
<tr>
<td><em>Azadirachta indica</em></td>
<td></td>
<td>B-Sitosterol (steroid)</td>
<td>Antihypoglycemic activity</td>
<td>86</td>
</tr>
<tr>
<td></td>
<td><img src="image" alt="Structure of B-Sitosterol" /></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Gymnema sylvestre R.Br</em></td>
<td></td>
<td>Gymnemic acids IV</td>
<td>Antihypoglycemic activity</td>
<td>87, 88</td>
</tr>
<tr>
<td></td>
<td><img src="image" alt="Structure of Gymnemic acids IV" /></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>
Apart from the above mentioned plants, the local Ayurvedic as well as the traditional Vaidyas, who trust primarily on herbal medicines, prescribe leaves of *Tabernaemontana coronaria* plant for treating various ailments. The plant is abundantly available in Shimoga district and the literature survey revealed that, so far no research reports available on anti-diabetic activity on this plant. This has provided plenty of scope for the research work to be carried out on *Tabernaemontana coronaria*. Hence a major research programme has been undertaken in our laboratory in this regard.
References


