CHAPTER 1
INTRODUCTION

1.1 DIABETES MELLITUS

*Diabetes* derives its source from a Greek word 'diabaino' which means to go through, and “mellitus” means sweet or sugar. Hence, the passing of sugar with urine may be the crude meaning of the word diabetes mellitus. The characteristic of Diabetes Mellitus is the lack of ability to control blood glucose, which finally passes through urine in excessive cases of diabetes mellitus. Diabetes Mellitus is a group of metabolic disorders characterized by abnormal high blood glucose (hyperglycemia) resulting from defects either in insulin secretion or insulin action or both *(David, 1996)*.

Due to lacking action of insulin on the target tissues, the metabolism of carbohydrates, fats and proteins is altered. Chronic hyperglycemia in diabetes is associated with damage of tissue, dysfunction and ultimate functional failure of various organs especially the eyes, kidneys, nerves, heart and blood vessels *(American Diabetes Association, 1998)*.

Several pathogenic processes are involved in the development of Diabetes Mellitus. Two major types of clinical syndromes due to increased hyperglycemia can be mentioned here. The first one is categorized by insulin dependent diabetes mellitus and onset in early age, with weight loss and ketonuria (passing of ketone bodies in urine). This is generally termed as type 1 or Insulin Dependent Diabetes Mellitus (IDDM). Fortunately the type 1 diabetes mellitus or (insulin dependent diabetes mellitus) prevalent in South East Asia including India as in the west. The second is
categorized by late onset and insensitivity to insulin deficiency. It is generally called Non-Insulin Dependent Diabetes Mellitus (NIDDM) or type 2 diabetes mellitus. The prevalence of diabetes, especially Non Insulin Dependent Diabetes Mellitus, is spiralling upwards, both in developed and developing countries. Fueled by fast economic growth, the prevalence of diabetes has now reached up to 8% of the world population. Weakly controlled diabetes aggravates the risk of diabetes complications and particularly cardiovascular diseases (Mayes, 1993). The adolescent people in India have Malnutrition Related Diabetes Mellitus (MRDM), which can be well controlled or reversed by proper and balanced diet. 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 (Wild et al., 2004). In 2008, an estimated 347 million people in the world had diabetes and the prevalence is rising, mainly in low- and middle-income countries. India had 69.2 million people living with diabetes (8.7%) as per the 2015 data. Of these, it remained undiagnosed in more than 36 million people (WHO, 2016).

In diabetes, although there is two to three fold increases in blood glucose concentration from the normal levels, the tissues themselves are starving of glucose. Apart from high glucose in blood, there is also an increase of fat and protein in the blood, which may build up. The body may break these down to get energy. The normal fasting blood glucose of humans is 60-100 mg/dl, which may increase up to 350 mg/dl in cases of diabetes. The functioning of the various organs is affected and the body function for maintenance of meostasis undergoes a long lasting change due to the increased level of the glucose concentrations in the blood. This have negative effect on the tissues which are dependent on insulin for glucose transport (namely
adipose, liver and muscle) and tissues which are independent of insulin for glucose transport like brain, kidney and red blood cells in different ways (Brownlee, 1986). The cause of Insulin Dependent Diabetes Mellitus has been reported recently to be T-cell mediated auto-immune towards glutamic acid decarboxylase (GAD).

1.2 PATHOPHYSIOLOGY

1.2.1 Type I Diabetes Mellitus (IDDM)

The autoimmune eradication of pancreatic β-cells, leads to a deficiency of insulin secretion which results in the metabolic derangements associated with Insulin Dependent Diabetes Mellitus. Further to the loss of insulin secretion, the function of pancreatic α-cells is also abnormal and there is extreme secretion of glucagons in Insulin Dependent Diabetes Mellitus. Normally, hyperglycemia leads to reduced glucagon secretion however, in patients with Insulin Dependent Diabetes Mellitus, glucagon secretion is not suppressed by hyperglycemia.

The resultant inappropriately elevated glucagon levels aggravate the metabolic defects due to insulin deficiency. The most pronounced example of this metabolic disorder is that patients with Insulin Dependent Diabetes Mellitus rapidly develop diabetic ketoacidosis in the absence of insulin administration. Even though insulin deficiency is the primary defect in Insulin Dependent Diabetes Mellitus, there is also a deficiency in the administration of insulin. There are several biochemical mechanisms that account for impairment of tissue’s response to insulin. Deficiency in insulin leads to unconstrained lipolysis and elevated levels of free fatty acids in the plasma, which suppresses glucose metabolism in peripheral tissues such as skeletal muscle. This impaired glucose utilization and insulin deficiency also reduces the
expression of a number of genes essential for target tissues to respond normally to insulin such as glucokinase in liver and the GLUT 4 class of glucose transporters in adipose tissue (Raju and Raju, 2010). Explained that the major metabolic derangements, which result from insulin deficiency in Insulin Dependent Diabetes Mellitus are impaired glucose, lipid and protein metabolism which are explained in details as follows.

1.2.1.1 Effect on glucose metabolism

Uncontrolled Insulin Dependent Diabetes Mellitus leads to increased hepatic glucose output. First, liver glycogen stores are mobilized then hepatic gluconeogenesis is used to produce glucose. Furthermore insulin deficiency impairs non hepatic tissue utilization of glucose. In particular in adipose tissue and skeletal muscle, insulin stimulates glucose uptake. This is consumate by insulin mediated movement of glucose transporters proteins to the plasma membrane of these tissues. Reduced glucose uptake by peripheral tissues in turn leads to a reduced rate of glucose metabolism. Moreover, the level of hepatic glucokinase is regulated by insulin. Therefore, a reduced rate of glucose phosphorylation in hepatocytes leads to increased delivery to the blood. Other enzymes concerned in anabolic metabolic metabolism of glucose are affected by insulin.

The mixture of increased hepatic glucose production and reduced peripheral tissues metabolism leads to elevated plasma glucose levels. While the capacity of the kidneys to absorb glucose is ensues the glycosuria. Glucose is an osmotic diuretic and an increase in renal loss of glucose is followed by loss of water and electrolyte. The result of the loss of water leads to the activation of the thirst mechanism (i.e.,polydipsia). The negative caloric balance, which results from the glycosuria and
tissue catabolism leads to an increase in the appetite and food intake that is polyphagia.

1.2.1.2 Effect on lipid metabolism

One major function of insulin is to stimulate the storage of food energy in the form of glycogen in hepatocytes and skeletal muscle, subsequent the consumption of a meal. Moreover, insulin stimulates hepatocytes to produce and store triglycerides in adipose tissue. In uncontrolled Insulin Dependent Diabetes Mellitus there is a rapid mobilization of triglycerides leading to increased levels of plasma free fatty acids. The free fatty acids are taken up by several tissues (except the brain) and metabolized to provide energy. In the absence of insulin, malonyl COA levels fall, and transport of fatty acyl COA into the mitochondria. Mitochondrial oxidation of fatty acids generates acetyl COA that can be more oxidized in the TCA cycle. However, in hepatocytes most of the acetyl COA is not oxidized by the TCA cycle but which is metabolized into the ketone bodies (acetoacetate and β-hydroxybutyrate). These ketone bodies are used for energy production by the brain, heart and skeletal muscle. In Insulin Dependent Diabetes Mellitus, the increased accessibility of free fatty acids and ketone bodies exacerbates the reduced utilization of glucose, which ensues the hyperglycaemia. Production of ketone bodies in excess of the body’s ability to utilize them leads to ketoacidosis. A spontaneous breakdown product of acetoacetate is the acetone which is exhaled by the lungs and which gives a distinctive odor to the breath. Normally, plasma triglycerides are acted upon by lipoprotein lipase (LPL) that requires insulin. LPL is a membrane bound enzyme on surface of the endothelial cells lining the vessels, which allows the fatty acids to be taken from circulating
triglycerides for storage in an adipocytes. The absence of insulin which results in the hypertriglyceridemia.

1.2.1.3 Effects on protein

Insulin regulates the synthesis of many genes, either positively or negatively, which is affecting the overall metabolism. Insulin has an overall effect on protein metabolism, increasing the rate of protein synthesis and decreasing the rate of protein degradation. Accordingly insulin deficiency will lead to increased catabolism of protein. The increased rate of proteolysis leads to the elevated concentration of amino acids in plasma (Raju and Raju, 2010). Glucogenic amino acids serve as precursors for hepatic and renal glyconeogenesis, which further contributes to the hyperglycaemia seen in Insulin Dependent Diabetes Mellitus (IDDM).
Figure 1.1. Glucose homeostasis. (While the blood glucose levels are increased, insulin is released from the beta cells and stimulates the liver and for the most other body cells to absorb glucose. The glucose is transformed to glycogen, triglycerides and protein. If the glucose levels are decreased, the secretion of glucagon from the alpha cells stimulates the liver to release glucose from the liver. Mainly the glucose is released from the breakdown of glycogen and the conversion of amino acids and fatty acids into glucose. Dysfunction in glucose metabolism results from deficiency in insulin (IDDM) and resistance to insulin action (NIDDM) by target tissues).

1.2.2 Type II Diabetes Mellitus (NIDDM)

Individuals with Non Insulin Dependent Diabetes Mellitus have measurable levels of circulating insulin, unlike patients with Insulin Dependent Diabetes Mellitus and the pathophysiology of type 2 diabetes mellitus (NIDDM) is described in Figure 1.1. On the basis of oral glucose tolerance testing the essential elements of type 2 diabetes mellitus (NIDDM) can be divided into four distinct groups:

- Those with normal glucose tolerance.
- Chemical diabetes (which is called impaired glucose tolerance).
- Diabetes with minimal fasting hyperglycemia (fasting plasma glucose less than 140 mg/dl).
- Diabetes mellitus in association with over fasting hyperglycemia (fasting plasma glucose greater than 140 mg/dl).

The individuals by means of impaired glucose tolerance have hyperglycemia inspite of having maximum levels of plasma insulin, signifying that they are resistant to the action of insulin. In the progression from impaired glucose tolerance to the diabetes mellitus, the level of insulin declines signifying that patients with type 2 diabetes
mellitus (NIDDM) have decreased insulin secretion. Insulin resistance and insulin deficiency are common in the average of type 2 diabetes mellitus (NIDDM) patients (Holt, 2004). Insulin resistance is the primary cause of type 2 diabetes mellitus (NIDDM), though some researchers compete that insulin deficiency is the primary reason because a moderate degree of insulin resistance is not enough to cause type 2 diabetes mellitus (NIDDM) (Raju and Raju, 2010). Generally most patients with the common form of type 2 diabetes mellitus (NIDDM) have both defects. Recent evidence has established a role for a member of the nuclear hormone receptor super family of proteins in the etiology of type 2 diabetes mellitus (NIDDM) (Raju and Raju, 2010).

Table 1.1: Differences between Type I and Type II diabetes mellitus.

<table>
<thead>
<tr>
<th></th>
<th>Type I diabetes mellitus</th>
<th>Type II diabetes mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology</strong></td>
<td>Autoimmune destruction of pancreatic β-cells</td>
<td>Insulin resistance with β-cell function to compensate</td>
</tr>
<tr>
<td><strong>Prevalance</strong></td>
<td>10%</td>
<td>90%</td>
</tr>
<tr>
<td><strong>Insulin levels</strong></td>
<td>Nil</td>
<td>Typically higher than normal</td>
</tr>
<tr>
<td><strong>Insulin action</strong></td>
<td>Nil</td>
<td>Decreases</td>
</tr>
<tr>
<td><strong>Insulin resistance</strong></td>
<td>Not part of syndrome, but may be present in obese patients</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Progress of disease</strong></td>
<td>Develops suddenly</td>
<td>Develops gradually and progresses slowly</td>
</tr>
<tr>
<td><strong>Life style</strong></td>
<td>Active or Sedentary</td>
<td>Sedentary</td>
</tr>
<tr>
<td><strong>Age of onset</strong></td>
<td>typically below 30</td>
<td>typically above 40</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>Faster onset of symptoms</td>
<td>Slower onset of symptoms</td>
</tr>
<tr>
<td><strong>Drug administration</strong></td>
<td>Insulin must be administered</td>
<td>Diet control and oral hypoglycemic agents often sufficient control</td>
</tr>
<tr>
<td><strong>Body weight</strong></td>
<td>Patients usually not overweight</td>
<td>Patients usually overweight</td>
</tr>
<tr>
<td><strong>Acute Complications</strong></td>
<td>Ketoacidosis, wasting</td>
<td>Hypoglycemia can lead to hyperosmotic seizures and coma</td>
</tr>
<tr>
<td><strong>Chronic complications</strong></td>
<td>Diabetic neuropathy</td>
<td>Diabetic neuropathy</td>
</tr>
<tr>
<td></td>
<td>Diabetic retinopathy</td>
<td>Diabetic retinopathy</td>
</tr>
</tbody>
</table>
1.3 COMPLICATIONS

1.3.1 Diabetic macro vascular complications

1.3.1.1 Coronary Heart Disease

Elderly adults with diabetes mellitus have a high incidence of Coronary Heart Disease (CHD). Using a composite definition of CHD (bypass surgery, angiographic evidence of coronary disease, previous percutaneous intervention and documented myocardial infarction (MI) or electrocardiographic features of MI), the prevalence among diabetes patients with a mean age of 80 years at an academic center was 44% (Ness et al., 1999). The prevalence of cardiovascular disease is higher in persons with diabetes mellitus compared to those without diabetes. An Italian cohort of 3,474 patients aged with 65–84 years old demonstrated the incidence of MI among patients with diabetes mellitus was 11.3%, among patients with impaired fasting glucose was 8.5%, and among patients with normal glycemia was 8%. The comparison from the diabetes mellitus group to the normal glycemia group was statistically significant ($P=0.032$). Older men with diabetes mellitus had a higher incidence of MI compared to women with diabetes mellitus (15.5% vs. 7.2%, $P=0.001$) (Motta et al., 2007), however, survival bias may be present. CHD among older patients with diabetes is a most important cause of mortality. The Cardiovascular Health Study included participants with aged 65 years old and older with and without diabetes. Compared to persons without diabetes, those with treated diabetes mellitus had an increased in amount of the risk of CHD related mortality (oral-treated diabetes mellitus: HR 2.47
95% CI 1.89–3.24 and insulin-treated diabetes mellitus: HR 2.75 95% CI 1.95–3.87) (Kronmal et al., 2006).

Several older diabetes patients have CHD cause of unawerness. An autopsy series included 293 persons with diabetes (mean age 73 years), without clinically known CHD. Nearly 75% of the patients had high grade coronary disease and greater than half had multivessel disease (Goraya TY et al., 2002). Silent ischemia is also a problem among the older patients with diabetes. A French study screened asymptomatic patients with diabetes for CHD. Their cohort included 130 patients with type 2 diabetes mellitus and a mean age of 60.7 years. With their screening method, 20.9% of asymptomatic men in this group had significant coronary lesions on angiography (Janand- Delenne et al., 1999).

1.3.1.2 Cerebrovascular Disease

Older adults with diabetes mellitus are at mainly high risk of morbidity and mortality from cerebrovascular disease (CVD). The incidence of CVD in the older population with diabetes is higher than those without diabetes mellitus. An Italian study investigated the incidence in a group of adults aged 65 to 84 years that were stratified into groups (diabetes, impaired fasting glucose, and normal glycemia). The incidence of CVD in patients with a history of diabetes mellitus was 10.6%. This was higher than the prevalence in patients without diabetes (7%, $P=0.003$). Looking specifically at the subgroup of patients aged 75 to 84, the prevalence among the patients with diabetes was 13% compared to 10% in patients without diabetes (Motta et al., 2007). Elderly patients with diabetes who experience excess worry related to diabetes mellitus symptoms, diet restrictions, treatment satisfaction, and medications and have
a lower sense of well being may be more probable to have a CVD. A prospective cohort study of 375 geriatric patients with a mean aged 75 years established that lower scores on the Geriatric Morale scale and Elderly Diabetes Burden scale were the predictors for CVD (HR=2.6, 95% CI=1.1–6.5, \( P=0.039 \)) \( \text{(Araki et al., 2004)} \). This suggests psychosocial factors may be associated with stroke events among elderly patients with diabetes.

### 1.3.1.3 Peripheral Vascular Disease

Peripheral Vascular Disease (PVD) is a common diabetes complication in older adults (aged older than 55 years). Among U.S. adults age 60 years and older, the incidence of PVD for patients with diabetes was almost twice as high compared to those without diabetes \( \text{(Kalyani et al., 2010)} \). A recent multicenter study estimated the prevalence of PVD to be 60.6% among a cohort study of 1,430 older adults diabetes patients aged 70 years and older (mean age 78 years). Predictors of an abnormal ankle brachial index (ABI) included male gender, smoking, dyslipidemia and having the other diabetes related complications \( \text{(Escobar et al., 2011)} \). A recent study in Malta also supports the finding that having other diabetes-related complications increases the risk of PVD. Investigators wanted to see what risk factors and complications associated with a low ABI in older patients with type 2 diabetes mellitus and proliferative retinopathy. The cohort's average age was 65 years with a mean duration of diabetes of 18.6 years. Their results show that dyslipidemia and vibration perception thresholds (neuropathy) were associated with low ABI. In addition, older age and declining GFR also were associated risk factors \( \text{(Magri et al., 2012)} \). Progressively older age increases the risk of developing PVD. An Indonesian study demonstrated that patients with type 2 diabetes mellitus age 70–80 years were
7.4 times more likely to develop PVD compared to patients with type 2 diabetes mellitus age 60–69 years (Kuswardhani and Suastika, 2010).

1.3.2 Diabetic micro vascular complications

1.3.2.1 Diabetic Retinopathy

Diabetic Retinopathy is a common microvascular complication of diabetes. An investigation of National Health and Nutritional Examination Survey (NHANES) data reports a crude prevalence of diabetic retinopathy at 29.5% among the patients age 65 years and older with diabetes. This incidence is similar to the prevalence of diabetes in patients age 40–64 years (28.0%, \( P=0.64 \) for comparison). Amongst the entire cohort of patients with diabetes, men were more probable than women to have diabetic retinopathy (31.6% vs. 25.7%, \( P=0.04 \)). Patients with retinopathy have a longer duration of diabetes (15 years vs 7.3 years, \( P<0.001 \)), higher HbA1c value (7.9% vs 7.05, \( P<0.001 \)), and were more probable to be using insulin (44.6% vs. 10.2%, \( P<0.01 \)) (Zhang et al., 2010). Retinopathy is less common among adults diagnosed with diabetes in older-age compared to middle-age (Selvin et al., 2006).

1.3.2.2 Diabetic Nephropathy

The diabetic nephropathy [Chronic kidney disease (CKD)] is a common diabetes related complication in older adults. For adults older than 60 years, the most common cause of CKD and end-stage renal disease (ESRD) in the United States is diabetic nephropathy (Rosner M et al., 2010). Among adults age 75 years and older, about 1/3 of new cases of ESRD are caused by diabetic nephropathy (Coresh et al., 2003). Comparing older adults with diabetes to those without, the incidence of CKD is consistently higher among patients with diabetes. This was demonstrated with a recent study of the Kidney Early Evaluation Program (KEEP) database (a
community-based screening program targeting the adults at high risk of kidney disease), NHANES data, and billing codes from a sample of the U.S. Medicare population. In all 3 databases sets, the incidence of CKD was higher in individuals older than 65 years diagnosed with diabetes compared to those without (KEEP 48.2% vs. 40.4%, NHANES 58.3% vs. 41.4%, Medicare 14.2% vs. 4.4%; \( P<0.001 \))  
\textit{(Stevens \textit{et al.}, 2010)}.

Over the last 25 years, the proportion of patients with diabetic nephropathy who have stage 5 chronic kidney disease and are initiated dialysis in the U.S. rose from one in six persons to almost one in two persons. These rising rates of diabetes-related ESRD correspond with the increased burden of diabetes. The older age population with diabetes remains a large proportion of those receiving dialysis for diabetic nephropathy (CKD). In Canada, this older diabetes population makes up 37% of the all patients receiving dialysis. This older diabetes population presents unique challenges in medical management. Most have multiple co morbid conditions, such as coronary heart disease and peripheral vascular disease. In the elderly, ESRD with diabetes is related with an increased risk of dementia which can further complicate therapy. Additionally, this older population may be more possible to have arteriovenous fistula complications. Amongst a cohort of patients 65 years and older, 28.6% of patients with diabetes had fistula failure compared to only 10.3% of patients without diabetes \( (P=0.04) \) \textit{(Lin \textit{et al.}, 1998)}. All of these comorbid conditions make caring for the elderly patient with diabetes and ESRD challenging \textit{(Coresh \textit{et al.}, 2003)}.
1.3.2.3 Diabetic Neuropathy

The distal sensorimotor poly neuropathy is one of the most common long-term complications of diabetes. Patients with diabetic peripheral neuropathy (DPN) are at a high risk for falling. For older patients with diabetes duration greater than 25 years, about half of them have comorbid DPN and this can be associated with functional impairment and decreased quality of life (Ghanavati et al., 2012). Factors associated with increased risk of DPN among the elderly with diabetes include female sex, longer duration of diabetes, retinopathy, stroke, hypertension, dyslipidemia, and a history of foot ulcers (Won et al., 2012).

As the severity of neuropathy increases, the functional impairment worsens and quality of life can be affected (Ghanavati et al., 2012). Examples of functional restrictions include reduced walking speed, cadence, and step length compared with patients without diabetes. Patients with DPN also display impaired peripheral sensation, reaction time, and balance (Menz et al., 2004).

1.4 ROLE OF ANTIOXIDANT IN DIABETES MELLITUS

Free radicals are reactive molecules created naturally in the human body during metabolic reactions. High levels of free radicals damages the cellular proteins, membrane lipids, nucleic acids and eventually leads to cell death. Free radicals are plays an important role in the pathogenesis of many chronic diseases including atherosclerosis, myocardial infarction, immune diseases and type 2 diabetes mellitus. Free radicals include the reactive oxygen species (ROS) and reactive nitrogen species (Tesfamariam, 1994).
In healthy subjects, an antioxidant compound counters the effects of free radicals (Wild et al., 2004). Antioxidants are characterized into two groups; enzymatic and non-enzymatic. Superoxide dismutase (SOD), Catalase (CAT), Glutathione reductase (GR), Glutathione peroxidase (GPx), Tyrodoxin reductase, Ariel esterase and para-oxonase are included in the enzymatic group and Vitamins A,C and E, Carotenoids, Glutathione, Flavonoids, other compounds such as alpha-lipoic acid, Co-enzyme Q10, Copper, Zinc, Magnesium and Selenium are all included in the non enzymatic group (Esteghamati et al., 2008).

Oxidative stress is defined as the increased generation of free radicals and/or impaired compensatory response of endogenous antioxidant defenses both are observed in the type 2 diabetes mellitus (Betteridge, 2000). Oxidative stress is a pathologic condition resulting from either increased production of free radicals decreased level of antioxidants. Hyperglycemia, by the promotion of lipid peroxidation of low density lipoprotein (LDL) can result in the production of free radicals (Maritim et al., 2003).

Diabetes is a metabolic disorder and is generally accompanied by the increased level of free radicals and decreased activity of antioxidants. Studies are shown that serum concentrations of SOD and other antioxidants such as vitamin E and α-lipoic acid are decreased in type 2 diabetes patients. There is evidence that deficiency of catalase (CAT) in erythrocytes is associated with increased risk of diabetes (Tesfamariam, 1994). There is considerable evidence that oxidative stress plays a key role in insulin resistance, impaired insulin secretion and many of the complications of diabetes such as micro-macro vascular damage (Ahmed, 2005).
1.5 PLANT PROFILE

Medicinal herbs are moving from fringe to mainstream use with a larger number of people seeking remedies and health approaches free from side effects. India has a gold mine of well-recorded and traditionally well-practiced knowledge of herbal medicine. Many medicinal plants are inferred as antidiabetic and antioxidant activities from ethanopharmacological evidences and no proper scientific studies are carried out and reported. Among these herbal resources, the medicinal plant of *Alysicarpus monilifer* is selected for the project work.

1.5.1 BOTANICAL DESCRIPTION (Shadma and Naheed, 2013)

Scientific Name: *Alysicarpus monilifer* (L.) DC.

Tamil name: Kasukkodi

Synonyms: *Hedysarum moniliferum* L.

Family: *Leguminosae*

Sub family: *Faboideae*

Tribe: *Desmodieae*

Sub tribe: *Desmodiinae*
1.5.2 Morphological description

Low growing, much branched, annual or perennial herb, 5–15 (–50) cm tall. Leaves simple; ovate, elliptical or lanceolate, cordate at the base, 2.5–7.5 cm long, prominently nerved, glabrous or sparsely pubescent beneath. Racemes spicate, axillary and terminal, 1–15 cm long; flowers lax to dense along racemes. Pods distinctly moniliform, 3- to 5-jointed, 1–2 cm long, calyx not longer than first joint; glabrous or sparsely pubescent; articles 2.5–3 mm long and 2–3 mm wide, with a smooth to reticulate surface sculpture.

1.5.3 Distribution


1.5.4 Uses/applications

Not tested, but could be a useful component of long-term and ley pastures in low to medium rainfall areas, especially under heavy grazing.

1.5.5 Ecology

1.5.5.1 Soil requirements

Grows on a wide range of soil types from deep sands and stony soils to cracking clays with a pH range of 5.5–8.

1.5.5.2 Moisture

Perennial types from India are found in areas with 600–1,500 mm annual rainfall, and annual types from Sudan in areas with 200–400 mm, and a short (<3 months) growing season.
1.5.5.3 Temperature

Mainly tropical lowlands (0–1,000 m asl) with average daily temperature range of 26–29°C. Perennial types are readily frosted but annuals because of early maturity largely avoid frost.

1.5.5.4 Light

No information available.

1.5.5.5 Reproductive development

At 21°S, flowering can occur at 60–70 days after establishment and seed can be mature at 90 days (annuals) and 110–115 days (perennials). Seed retention is poor with moniliform pods readily disarticulating between articles. A high percentage (>70%) of seed is hard at maturity. Flowers August - October (- November) at 21°N in India.

1.5.5.6 Defoliation

Prostrate types should tolerate intensive grazing in permanent pastures while the more erect forms are likely to be less tolerant of grazing and may be more useful in forage or as leys in cropping systems.

1.5.5.7 Fire

Response to fire in the vegetative state is not known but heavy and early seeding should minimise the chances of loss from pastures that are inadvertently burnt.

1.5.6 Agronomy

Guidelines for the establishment and management of shown pastures.

1.5.6.1 Establishment

Moderate seed size and a relatively fast germination rate give a quick and reliable establishment. Some form of seed scarification to break hard seed may be
necessary. Not highly competitive with weeds or established perennial pasture plants.

1.5.6.2 Fertiliser

Could be expected to respond to P, K and S on soils that are deficient or below the optimum pH range of 6–8.

1.5.6.3 Compatibility (with other species)

Appears best suited in combination with less vigorous grasses. Heavy grazing or extreme dry seasons that set back companion grasses may enhance legume persistence and spread. Seedling regeneration has been successful at low to medium rainfall sites in a sub-humid environment on the tropic where competition from grass was not extreme.

1.5.6.4 Companion species

Grasses: In India, *A. monilifer* is often found with *Bothriochloa*, *Dichanthium*, *Chrysopogon* and *Heteropogon* on rocky or eroded areas where grazing is heavy.

1.5.6.5 Pests and diseases

No information available.

1.5.6.6 Ability to spread

Early seeding and probable ability to withstand heavy grazing could result in spread from sown areas.

1.5.6.7 Weed potential

Low growth habit limits its ability to dominate companion species.

1.5.7 Feeding value

1.5.7.1 Nutrition value

No information available.
1.5.7.2 Palatability/acceptability
Well eaten by cattle.

1.5.7.3 Toxicity
No record of toxicity.

1.5.8 Production potential

1.5.8.1 Dry matter
Low to moderate yields in experimental sowings in Queensland, Australia.

1.5.8.2 Animal production
No information available.

1.5.9 Genetics/breeding
No information available

1.5.10 Seed production
Free seeding and relatively large seed, but would be difficult to harvest because of low growing habit and disarticulation of pod articles.

1.5.11 Herbicide effects
No information available.

1.5.12 Strengths
- Easily established.
- High palatability.

1.5.13 Limitations
Low DM yield in mixed pastures.