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Potential use of Sugarcane leaves in the management of Diabetes mellitus

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Abstract

Sugarcane has a historical use in the treatment of various conditions, including jaundice, hemorrhage, urinary disorders, gastrointestinal disturbances, and ocular diseases. The plant is also recognized for its cardiotonic, diuretic, and laxative properties. It exhibits anti-inflammatory, antimicrobial, anti-ulcerative, and antioxidant activities. Policosanol, a key constituent, is known to lower cholesterol levels by inhibiting HMG-CoA reductase, a critical enzyme in cholesterol biosynthesis.

Diabetes mellitus is a metabolic disorder characterized by the body's inability to effectively regulate blood glucose levels. The pancreas, normally responsible for insulin production, fails to produce sufficient quantities or the body develops insulin resistance. This leads to hyperglycemia.

Fatty acids contribute to an increase in diacylglycerol, triglycerides, ceramides, and reactive oxygen species, ultimately leading to mitochondrial dysfunction, endoplasmic reticulum stress, and impaired autophagy. These factors contribute to beta-cell dysfunction and death, culminating in the development of type 2 diabetes. Given the cholesterol-lowering properties of policosanol, sugarcane leaves may offer potential benefits in management of type 2 diabetes.

Keywords: Sugarcane; Leaves; Policosanol; Antioxidant; Anti-inflammatory; Antidiabetic

1. Introduction

Diabetes is a condition where the body cannot effectively process food for energy. Most foods contain glucose, a type of sugar, which the body uses as fuel. The pancreas, an organ near the stomach, produces a hormone called insulin to help glucose enter the body's cells. In people with diabetes, either the body does not produce enough insulin or cannot use it properly. This causes blood sugar levels to rise, which is why diabetes is often referred to as "sugar" [1].

Diabetes is a condition affecting how the body processes carbohydrates. It is characterized by elevated blood sugar levels (hyperglycemia) and excess glucose in the urine (glycosuria) [2]. Diabetes Mellitus (DM) is a metabolic condition characterized by persistently high blood sugar levels. It also involves impaired processing of carbohydrates, fats, and proteins in the body [3]. High blood sugar levels (hyperglycemia) associated with diabetes can damage small and large blood vessels. This can result in eye problems, including blindness, kidney disease, and heart attacks [4]. Diabetes mellitus is diagnosed when blood sugar levels are abnormally high due to insufficient insulin or the body's resistance to insulin [5]. Insulin is a hormone produced by the pancreas that controls blood sugar levels by regulating the body's glucose production and storage [6].

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Other factors contributing to diabetes include the destruction of insulin-producing cells in the pancreas, leading to insulin deficiency. The abnormal processing of carbohydrates, fats, and proteins occurs due to a lack of insulin's effectiveness (WHO, 2015). Diabetes is a long-term condition that can be managed but not cured [7]. In 2014, approximately 9% of adults aged 18 and over worldwide had diabetes. Diabetes caused an estimated 1.5 million deaths in 2012. Over 80% of these deaths occurred in low and middle-income countries. The World Health Organisation (WHO) predicts diabetes will be the seventh leading cause of death in 2030 [8].

The chronic disease, DM is of three types:

- Type 1
- Type 2
- Type 3

Type 1 diabetes, also known as insulin-dependent diabetes mellitus (IDDM), is a condition where the body produces little or no insulin. This occurs due to a decrease in insulin secretion by the body's cells. It affects between 5% and 10% of all diagnosed diabetes cases.

Type 2 diabetes, also known as non-insulin-dependent diabetes mellitus (NIDDM), is a condition where the body either doesn't produce enough insulin or doesn't use insulin effectively [9]. This type of diabetes is primarily caused by two issues: the pancreas's inability to produce sufficient insulin and the body's resistance to the insulin that is produced [10]. There is a strong genetic link to Type 2 diabetes, and it develops due to a combination of inadequate insulin production and the body's tissues becoming less responsive to insulin. As a result, individuals with Type 2 diabetes have a relative insulin deficiency. This is the most common form of diabetes, affecting between 90% and 95% of people with the condition.

Type 3 Gestational diabetes mellitus (GDM) occurs when a woman's body is unable to produce enough insulin to manage the increased demands of pregnancy. This condition affects between 2% and 5% of pregnant women [11]. **Gestational diabetes mellitus (GDM)** is a condition where the body becomes less tolerant to carbohydrates, leading to high blood sugar levels that first appear or are recognised during pregnancy [12]. It is associated with the most common metabolic complications of pregnancy and increased risks of fetal death and illness [13].

Diabetes is treated with medications such as sulfonylureas (including glimepiride, glibenclamide, and glipizide), metformin, biguanides, thiazolidinediones, tolbutamide, and alpha-glucosidase inhibitors (like acarbose and miglitol). Persistently high blood sugar levels can lead to both large and small blood vessel damage, increasing the risk of heart disease, including atherosclerosis, and kidney failure. Oxidative stress contributes to tissue damage, including damage to the retina and ulcers, through a process called lipid peroxidation [14,15]. Other complications associated with diabetes include kidney disease, nerve damage, high blood pressure, and foot ulcers. Some common side effects of anti-diabetic medications are weight gain, itching, fatigue, digestive problems, and swollen legs [8,16].

Sugarcane, a plant belonging to the grass family (Poaceae), is a significant global crop due to its economic and medicinal value. It is composed of roots, stalks, leaves, and flowers. Chemically, sugarcane contains substances such as fatty acids, alcohols, phytosterols, flavonoids, alkaloids, glycosides, and phenolic acids. The juice extracted from sugarcane has been traditionally used to treat conditions including jaundice, bleeding, difficulty urinating, and lack of urine production [17].

Sugarcane has been reported to possess antimicrobial, ulcer-healing, anti-inflammatory, and antioxidant properties [18-20]. Sugarcane leaves are also used as fodder for animals that chew cud, such as cows and sheep [21]. There has been a significant rise in the global popularity of herbal products due to the belief that they are effective with minimal side effects. Pharmaceutical companies are conducting extensive research into plants to identify potential new medicines [18-20]. Sugarcane juice is commercially extracted on a large scale. The leaves, often discarded as waste, may hold untapped medicinal properties. While sugarcane leaves are widely used as animal feed, their potential to treat diabetes has yet to be explored.

2. Diabetes mellitus

Diabetes is the state in which the body does not accurately process food for use as energy. Most of the food we eat is changed into glucose, or sugar, for our bodies to use for energy. The pancreas, an organ that presents near the stomach, makes a hormone called insulin to help glucose get into the cells of our bodies. When someone has diabetes, his or her body either doesn't make sufficient insulin or can't use its own insulin as well as it should. This motivates sugars to build up in your blood. This is why many people refer to diabetes as “sugar” [1]. Diabetes is known as a disorder of carbohydrates metabolized characterized by high blood sugar level (hyperglycemia) and high levels of glucose in urine (glycosuria) [2]. Diabetes Mellitus (DM) is a metabolic disorder characterized by the presence of chronic hyperglycemia accompanied by greater or lesser impairment in the metabolism of carbohydrates, lipids and proteins [3].

Diabetes is a condition where the body cannot effectively regulate its blood sugar levels. People with diabetes have high blood sugar, also known as hyperglycemia. This occurs due to problems with insulin, a hormone that helps the body's cells absorb glucose for energy. Diabetes develops when the body either doesn't produce enough insulin or can't use it properly [22]. Diabetes was first recognised as a condition linked to excessive thirst, frequent urination, and weight loss in ancient times. High levels of blood sugar (hyperglycemia) cause excess sugar to be released in the urine. The hormone insulin controls blood sugar levels. In diabetes, either the body doesn't produce enough insulin or can't use it effectively, leading to hyperglycemia [7]. Hyperglycemia simply means high blood sugar levels. This condition can damage both large and small blood vessels, leading to serious health problems such as vision loss, blindness, kidney disease, and heart attacks [4].

Diabetes is a complex condition influenced by both genetic and environmental factors**.** Genetic factors can affect how the body produces or responds to insulin, while lifestyle factors such as obesity, lack of exercise, and stress can also contribute. Ultimately, diabetes is diagnosed when blood sugar levels are consistently high due to insufficient insulin or the body's resistance to insulin [5,23]. Insulin is a hormone produced by the pancreas, managing the blood glucose level by regulating the production and storage of glucose. Insulin is a protein constitute of 2 polypeptide chains A (with 21 amino acid residues) and B (with 30 amino acid residues). Chains A and B are joined by disulphide bridges. Insulin is synthesized in the beta cells of pancreas [6,24].

2.1. Types of diabetes mellitus

- **Type 1** is insulin-dependent diabetes mellitus (IDDM), in which the body does not produce any insulin or lack of insulin. There is destruction of β –cells due to which insulin secretion decreases. Type 1 diabetes mellitus is an inveterate autoimmune disease affiliated with selective destruction of insulin-producing pancreatic β-cells. The onset of clinical disease represents the end stage of β-cell downfall leading to type 1 diabetes mellitus. It is characterised by ketoacidosis (accumulation of ketone bodies). It most often takes place in children and young adults. It is also entitled as juvenile-onset diabetes. Type 1 diabetes accounts for 5–10% of population [25]. Heredity plays an important part in the development of type 1 diabetes [26].
- **Type 2** is non-insulin-dependent diabetes mellitus (NIDDM), in which the body does not produce enough, or improper use of secreted insulin. Insulin secretion reduces with gradual beta cell failure. There are two main pathological defects in type 2 diabetes which are impaired insulin secretion through a defunction of the pancreatic β-cell, and impaired insulin action through insulin resistance [27]. Type 2 diabetes is nearing endemic proportions, due to an increased number of elderly people, and a greater prevalence of obesity and sedentary lifestyles. It is also entitled as adult-onset diabetes. Type 2 DM is the most common form of the disease accounting for 90–95% of diabetes.
- **Type 3** is Gestational diabetes mellitus (GDM), occurs when a woman's pancreatic function is not sufficient to overcome the diabetogenic environment of pregnancy. Pregnancy confers an event of insulin resistance and hyperinsulinemia that may animate some women to develop diabetes. GDM is represented as glucose intolerance that was not present or known prior to pregnancy. GDM affects between 2% -5% of pregnant women [11]. In pregnancy, mothers need to make 2 to 3 times the normal amount of insulin. The placenta (the blood source for the baby) produces hormones that help the baby grow and develop. Some of these hormones block the action of the mother's insulin which is called insulin resistance. During pregnancy, if the body is unable to produce the extra insulin more resistant gestational diabetes develops [28]. **Gestational diabetes mellitus (GDM)** is a condition that develops during pregnancy when a woman's body becomes less tolerant to glucose, leading to high blood sugar levels. It is a common pregnancy complication linked to increased risks for both mother and baby. Factors such as impaired glucose tolerance and certain risk factors can contribute to the development of GDM [12-14].

2.2. Clinical symptoms of diabetes mellitus

- Hyperglycaemia
- Glycourea (Glycogen in urine)
- Polyurea (excess urine excretion)
- Polydypsia (excess thirst)
- Polyphagia (excess appetite)
- Ketoacidosis

2.3. Pathophysiology of diabetes mellitus

- **Type 1 diabetes** is a condition where the body produces very little or no insulin. Insulin is a hormone essential for regulating blood sugar levels. In this type of diabetes, the pancreas's insulin-producing cells (beta cells) are destroyed by the immune system. This destruction prevents the pancreas from responding to signals that normally stimulate insulin production. As a result, blood sugar levels rise, and the body breaks down fat for energy, leading to a condition called ketosis. People with type 1 diabetes require insulin injections to manage their blood sugar levels. The exact cause of type 1 diabetes is unknown, but it is believed to involve a combination of genetic susceptibility and environmental factors. The immune system mistakenly attacks the body's own pancreatic beta cells, leading to their destruction. This autoimmune process is often preceded by the presence of antibodies against pancreatic cells or insulin [7].
- **Type 2 diabetes** is a complex condition influenced by both genetic and lifestyle factors. These factors include a family history of diabetes, obesity, poor diet, lack of exercise, and ageing. The main characteristics of type 2 diabetes are the body's reduced ability to produce insulin (insulin secretion) and its decreased responsiveness to insulin (insulin resistance). Insulin resistance is often the primary factor leading to the development of the condition. Over time, the pancreas's ability to produce insulin gradually declines [25].
- **Both type 1 and type 2 diabetes are characterised by a gradual decline in the function of insulin-producing cells (beta cells).** Cell death, or apoptosis, is a primary cause of this decline. While the specific triggers for cell death differ between the two types, both involve the activation of inflammatory molecules like interleukin-1 beta and nuclear factor-kappa B. Ultimately, this leads to impaired glucose control, which is a hallmark of type 2 diabetes [29].

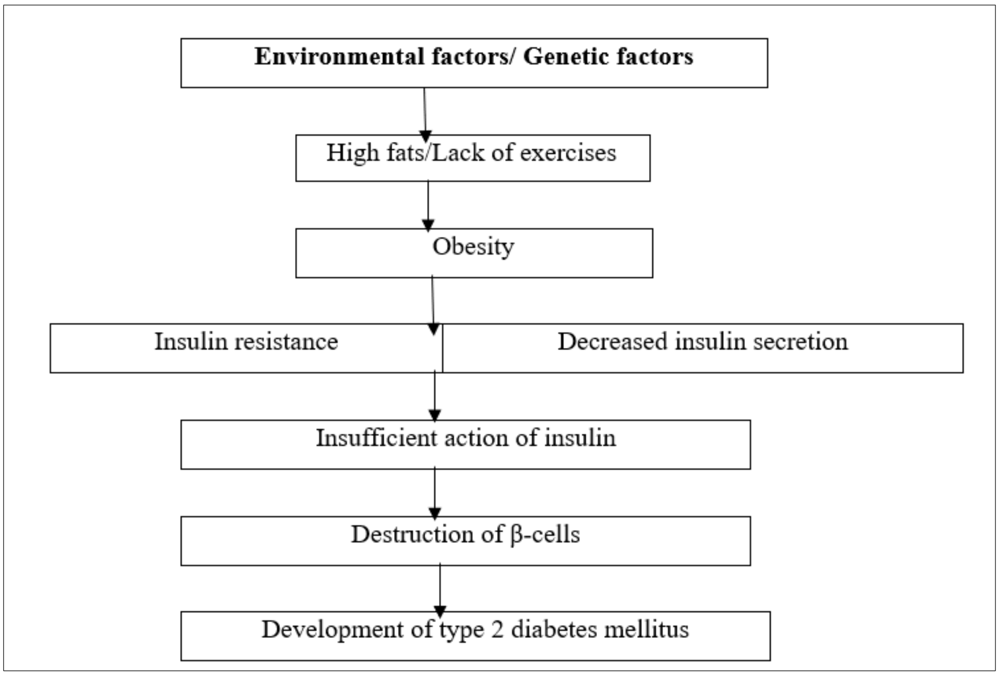


Figure 1 Schmatic representation of etiology of type 2 diabetes mellitus

Insulin at the cellular level involves two processes. First, insulin must bind to a specific receptor on the cell surface. Second, this interaction is followed by a series of intracellular events, including enhanced glucose transport into the cell and stimulation of a variety of intracellular enzymatic pathways [30]. The decrease in insulin binding is the result of a decrease in total insulin-binding capacity without alteration in insulin-binding affinity. Disturbance in hepatic glucose production, a post receptor defect will emerge and eventually become the dominant defect responsible for the insulin resistance [31]. β-cell mass is decreased in type 2 diabetes and that mechanism underlying this is increased β-cell apoptosis. Since the major defect leading to a decrease in β-cell mass in type 2 diabetes is increased apoptosis, while new islet formation and β-cell replication are normal [32].

Free fatty acids metabolites activate a serine kinase cascade, which leads to defects in insulin signaling downstream to the insulin receptor. The complex network of adipokines released from adipose tissue modulates the response of tissues to insulin. The insulin receptor substrate-2, the protein kinase B and the forkhead transcription factor Foxo 1a dysfunction of these proteins results in insulin resistance [33].

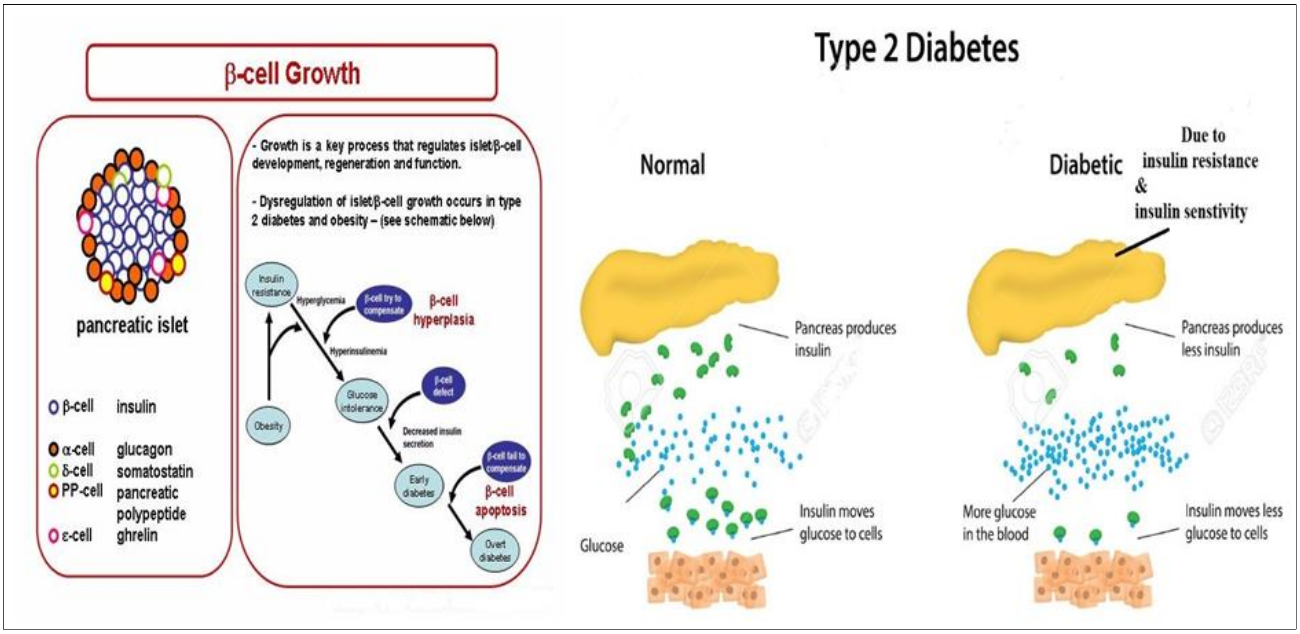


Figure 2 β-cell dysfunctioning in type 2 diabetes mellitus [33]

3. Mechanism of diabetes mellitus

3.1. Role of Oxidative stress in diabetes

Oxidative stress plays a role in the development of diabetes by affecting both insulin production and the body's response to insulin. The process involves an increase in reactive oxygen species (ROS), which are harmful molecules. Additionally, the activity of nitric oxide synthase, an enzyme involved in the production of nitric oxide, is linked to the development of diabetes [34-36]. In diabetes, the body produces harmful molecules called free radicals. These are generated through various processes including the breakdown of sugars and fats. Free radicals damage cells, including those involved in insulin production and function. This damage contributes to insulin resistance and the overall development of diabetes. Additionally, people with diabetes often have lower levels of antioxidants, which normally protect against these harmful molecules. This imbalance between free radicals and antioxidants, known as oxidative stress, is a key factor in the progression of type 2 diabetes [37,38].

3.2. Role of Mitochondrial dysfunction

Elevated glucose levels (hyperglycemia) trigger a series of enzyme-driven reactions within mitochondria. These reactions involve the activation of NADPH oxidase, the disruption of nitric oxide synthases, and the stimulation of xanthine oxidase. Additionally, glycated proteins can contribute to the generation of reactive oxygen species (ROS). These findings indicate that multiple factors may lead to excessive ROS production and oxidative stress in diabetes. Mitochondria are believed to be the primary source of ROS in this condition [38,39]. In individuals with diabetes, the protein composition of the inner mitochondrial membrane is reduced, and alterations in mitochondrial protein production occur. Mitochondrial dysfunction can be triggered by a combination of high glucose and fatty acid levels (glucolipotoxicity) as described by Youle et al. (2012). A persistent imbalance between excessive production of reactive oxygen species (ROS) and insufficient antioxidant defenses results in oxidative stress, which can ultimately damage mitochondria and lead to the death of beta cells [40,41].

3.3. Role of Endoplasmic reticulum stress

Endoplasmic reticulum (ER) stress can trigger beta cell activation. This stress, caused by high levels of glucose and fatty acids (glucolipotoxicity) and inflammatory substances, can lead to beta cell dysfunction. The ER is crucial for protein folding, quality control of newly produced proteins, and lipid synthesis. Protein production takes place within the ER and is vital for protein translation. The response of the pancreas to these challenges varies between individuals based on their genetic makeup, but may include inflammation, ER stress, metabolic and oxidative stress, protein aggregation, and damage to islet cells. Without treatment, these interconnected stressors worsen over time, resulting in impaired beta cell function (accompanied by increased glucagon production) and ultimately type 2 diabetes [42-44].

Beta cell death (apoptosis) occurs through several mechanisms, including an increase in ceramides, the activation of caspases, decreased levels of Bcl-2 protein, inflammation, reactive oxygen species (ROS) production, and the accumulation of unfolded proteins. Saturated fatty acids contribute to beta cell apoptosis, while unsaturated fatty acids generally have a protective effect. These fatty acids induce endoplasmic reticulum (ER) stress primarily by disrupting protein processing, transport, and calcium regulation [45].

3.4. Role of Islet inflammation

Prolonged exposure of pancreatic islet cells to high blood sugar levels (hyperglycemia) leads to increased levels of saturated fatty acids and reactive oxygen species (ROS). This can trigger the production of inflammatory substances such as tumor necrosis factor-alpha (TNF-α), nuclear factor kappa B (NF-κB), and interleukin-1 beta, ultimately causing beta cell dysfunction and death [46]. Inflammatory substances such as interleukin-1 beta (IL-1β) and interferon-gamma (IFN-γ) can also induce endoplasmic reticulum stress. Continuous activation of the nuclear factor kappa B (NF-κB) can lead to impaired cell function [47,48].

3.5. Role of Lipid induced β-cells failure in type 2 diabetes mellitus

Fatty acids can trigger the production of harmful substances such as diacylglycerol, triacylglycerol, ceramides, and reactive oxygen species. These lipid molecules initiate a series of damaging processes within beta cells, including endoplasmic reticulum stress, mitochondrial dysfunction, and impaired cellular waste removal (autophagy). These factors contribute to beta cell failure and programmed cell death (apoptosis), ultimately leading to the development of type 2 diabetes [49].

Obesity is influenced by both genetic and environmental factors. It is strongly linked to the development of type 2 diabetes [50]. Research suggests that obesity is not simply a risk factor, but a direct cause of type 2 diabetes in people with a genetic predisposition [51]. People with type 2 diabetes often have excess fat around their upper body. Additionally, the types of nutrients consumed have also been identified as a risk factor for developing type 2 diabetes [52].

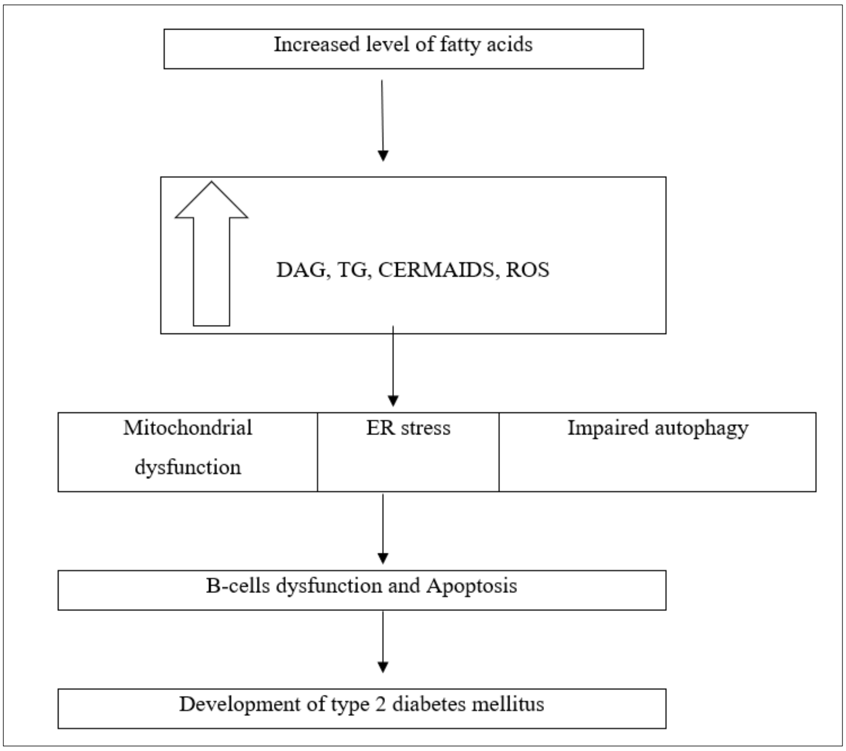


Figure 3 Lipid-induced b-cell failure in type 2 diabetes

4. Sugarcane: plant description

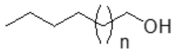
Sugarcane (Saccharum officinarum Linn) is a long-established energy source for humans, classified as a perennial grass within the Poaceae family. Originating from tropical South and Southeast Asia, sugarcane is a major crop in India.

Its name stems from the Greek word 'sakcharon', meaning sugar, due to its high sucrose content. Characterized by thick, unbranched stalks growing in clumps, sugarcane can reach heights of up to five metres. Underground rhizomes produce new shoots. The plant has long, serrated leaves and a feathery, pinkish flower head. The fruit is a small, dry seed. The sugarcane plant typically measures three to five metres in height and approximately five centimetres in diameter. Its sweet taste is a result of its high sucrose concentration. The stems, which can be green, pinkish, or purple, contain a fibrous white interior filled with sugary sap. The elongated leaves have thick midribs and serrated edges, reaching lengths of 30 to 60 centimetres and widths of about five centimetres. The plant produces a panicle-shaped inflorescence up to 60 centimetres long, with small, dry fruits containing a single seed [17].

Figure 4 Sugarcane

4.1. Phytochemistry

4.1.1. Phyto-chemistry of sugarcane wax



N=20 – Tetracosanol (2) ($C_{24}H_{50}O$)	n =21 – Hexacosanoicacid (10) ($C_{26}H_{52}O_2$)
N= 22 – Hexacosanol (3) (C26H54O)	n=22 – Heptacosanoicacid (11) ($C_{27}H_{54}O_2$)
N= 23 – Heptacosanol (4) ($C_{27}H_{56}O$)	n =23 –Octaacosanoicacid (9) ($C_{28}H_{56}O_2$)
N=24 – Octacosanol (1) ($C_{28}H_{58}O$)	n=24 – Nonacosanoicacid (12) ($C_{29}H_{56}O_2$)
N=25 – Nonacosanol (5) ($C_{29}H_{60}O$)	n=25 – Triacontanoicacid (13) ($C_{30}H_{60}O_2$)
N=26 – Triacontanol (6) ($C_{30}H_{62}O$)	n=26 – Hentriacontanoicacid (14) ($C_{32}H_{62}O_2$)
N=28 – Dotriacontanol (7) ($C_{32}H_{66}O$)	n= 27 – Dotriacontanoicacid (15) ($C_{32}H_{64}O_2$)
N=30 – Tetratriacontanol (8) ($C_{32}H_{70}O$)	n =28 – Tritriacontanoicacid (16) ($C_{33}H_{66}O_2$)

Figure 5 (a) Long chain saturated fatty alcohols, (b) Long chain saturated fatty acids present in D-003

Sugarcane wax is a powdery substance, ranging in colour from white to dark yellow, found on the surface of sugarcane stalks and leaves. It forms a protective layer similar to a cuticle. Given its widespread use in industry, cosmetics, and pharmaceuticals, sugarcane wax is a significant component to consider when analyzing the chemical composition of sugarcane. The wax content of sugarcane typically ranges from 0.1 to 0.3% [54,55]. Sugarcane wax is a valuable commercial resource for extracting long-chain fatty compounds, including alcohols, acids, esters, aldehydes, and ketones. Additionally, it contains policosanols, D-003, and other substances like steroids and terpenoids. Policosanols are a group of long-chain alcohols, with octacosanol being the most abundant component, making up between 50 and 80% of the total policosanol content [55]. Sugarcane wax also contains significant quantities of long-chain fatty acids, which are known collectively as D-003. These fatty acids are present in smaller amounts compared to the alcohols. In addition to these components, sugarcane wax has been found to contain various phytosterols, steroids, and other complex compounds derived from terpenes [14,48,56].

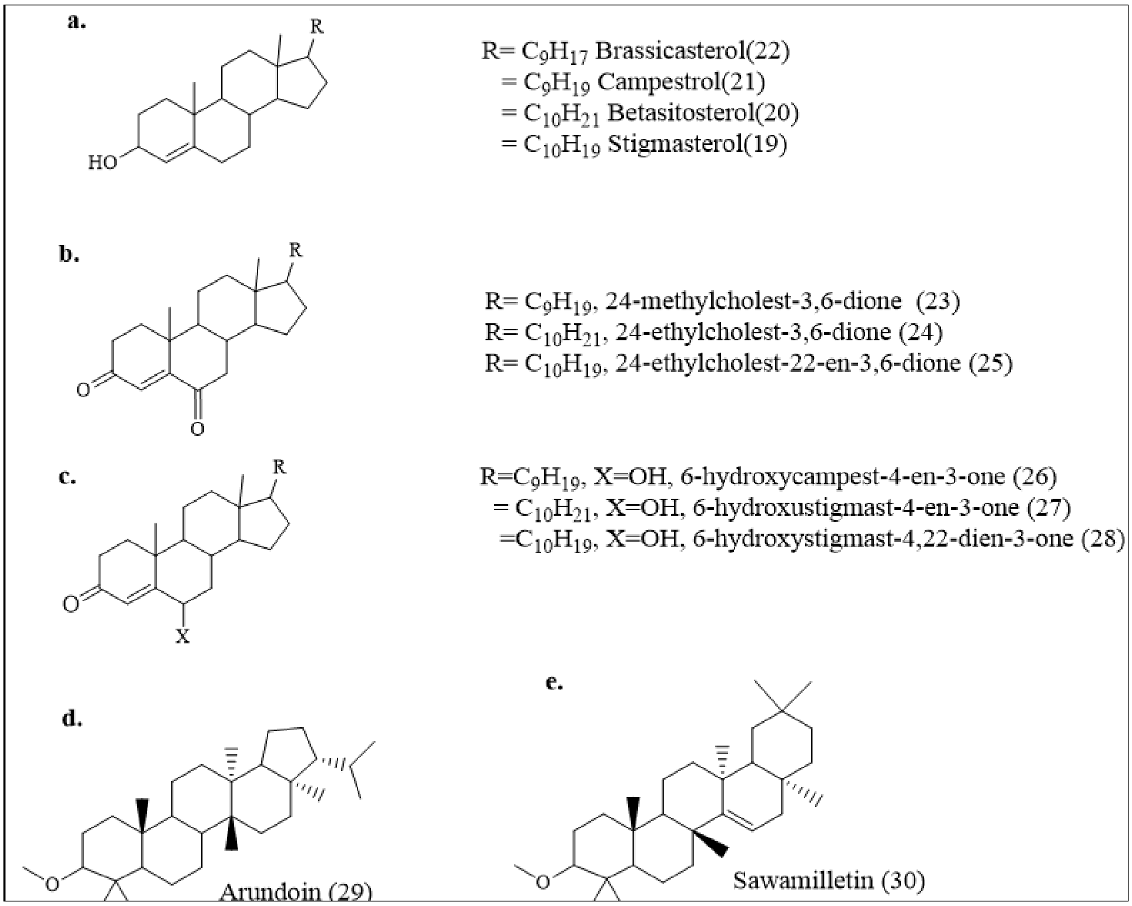


Figure 6 (a) Simple phytosterols; (b) Ketosteroids; (c) Hydroxy ketosteroids; (d) Higher terpenoids

4.1.2. Phyto-chemistry of sugarcane juice

Sugarcane juice contains substances such as chlorogenic acid, cinnamic acid, and flavones. The colour components found in sugarcane juice can be categorized into four main groups: plant pigments, polyphenolic compounds, caramels, and breakdown products formed from the interaction of sugars and amino acids [57,58]. Analysis of sugarcane juice using HPLC-DAD identified several phenolic compounds, including hydroxycinnamic acid, sinapic acid, and caffeic acid, as well as flavones like apigenin, luteolin, and tricin. Four previously unrecorded flavones, swertisin, tricin-7-O-neohesperoside-4'-O-rhamnoside, tricin-7-O-methylglucuronate-4'-O-rhamnoside, and tricin-7-O-methylglucuronide, were discovered and identified in sugarcane juice. In addition, several new types of flavone glycosides containing acyl groups were isolated, including tricin-7-O-β-(6'-methoxycinnamic)-glucoside, luteolin-8-C-rhamnosyl glucoside, and tricin-4'-O-(erthroguaicylglyceryl)-ether, alongside the known compound orientin [59,60].

4.1.3. Phyto-chemistry of sugarcane leaves

Sugarcane leaves are another significant source of policosanols and D-003 due to their protective wax coating. These leaves also contain phenolic compounds, including flavonoids. Through a detailed analysis of a methanol-based extract of sugarcane leaves, several flavonoid compounds were identified, both those attached to sugar molecules (O-glycosides) and those with a carbon-sugar bond (C-glycosides). These included diosmetin-8-glucoside, orientin, tricin-7-O-neoheperoxide, vitexin, luteolin-8-C-rhamnosyl glucoside, and tricin-4-O-ether [60,61].

4.1.4. Phyto-chemistry of sugarcane products

The chemical composition of various sugarcane products, including mill syrups, brown sugar, molasses, and non-centrifugal sugar, has been extensively researched. Three previously unknown flavonoid glycosides, tricin7-(2'-rhamnosyl)-α-galacturonide, orientin-7, 3'-dimethyl ether, and iso-orientin-7,3'-O-dimethyl ether, were discovered and identified in mill syrups (Mabry TM et al., 1984). Additionally, a new type of O-glycoside compound, dehydroconiferylalcohol-9'-O-β-D-glucopyranoside, was isolated from sugarcane molasses [62,63].

4.2. Pharmacological activities

Studies have shown that extracts from sugarcane peel have demonstrated an anti-proliferative effect, meaning they can inhibit the growth of cells, with the strength of this effect increasing as the concentration of the extract is raised [64]. Sugarcane leaves have anti-inflammatory properties [19]. Sugarcane juice is abundant in antioxidants and has been shown to effectively safeguard plasmid DNA and improve the survival rate of E. coli bacteria when exposed to radiation [65]. Sugarcane leaves have the ability to protect against ulcers [19]. Policosanol, derived from sugarcane wax, has been found to prevent the formation of atherosclerotic plaques [66]. Extracts from sugarcane leaves using chloroform were found to be effective against all tested microorganisms [18]. Sugarcane jaggery is believed to help regulate blood sugar levels (anti-diabetic). Policosanol, a compound found in sugarcane, shows promise as a natural alternative to traditional cholesterol-lowering medications. It has been shown to reduce overall cholesterol levels and levels of 'bad' cholesterol (LDL) in the blood. Additionally, policosanol from sugarcane wax is effective and well-tolerated in managing high cholesterol and type 2 diabetes. Research has also demonstrated that a compound derived from sugarcane juice, known as tricin, possesses antioxidant and anti-growth properties [20,60,67,68].

5. Conclusion

Sugarcane juice has been traditionally used to treat jaundice, hemorrhage, difficulty urinating, and complete lack of urine. It is reported to possess anti-inflammatory, antimicrobial, ulcer-preventative, and antioxidant properties. Additionally, sugarcane leaves serve as fodder for livestock.

Sugarcane contains a variety of plant compounds, including saturated fats, flavonoids, alkaloids, glycosides, tannins, triterpenoids, and long-chain alcohols. Notably, sugarcane leaves are a rich source of policosanols and a substance known as D-003, both found within the plant's protective wax coating. These leaves also contain phenolic compounds, such as flavonoids.

Policosanol, a compound derived from sugarcane, shows potential as a natural alternative to cholesterol-reducing medications. Studies indicate it can lower overall cholesterol levels and levels of 'bad' cholesterol (LDL) in the blood. Furthermore, policosanol extracted from sugarcane wax is effective and well-tolerated in managing high cholesterol and type 2 diabetes.

Elevated levels of fatty acids contribute to an increase in diacylglycerol, triglycerides, ceramides, and reactive oxygen species, ultimately leading to mitochondrial dysfunction, endoplasmic reticulum stress, and impaired autophagy. These factors contribute to beta-cell dysfunction and death, culminating in the development of type 2 diabetes. Given the cholesterol-lowering properties of policosanol, sugarcane leaves may offer potential benefits in management of type 2 diabetes. Yet further Pharmacological research is needed to be done for better understanding of the mechanism of action of this phytochemical.

Compliance with ethical standards

Disclosure of conflict of interest

There is no conflict of interest between authors.

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