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Antidiabetic Potential Orchids: A Promising Avenue for Diabetes Mellitus Management

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ABSTRACT

Diabetes mellitus is a widespread chronic metabolic disorder which affecting millions of individuals globally. A common yet complex and challenging condition like diabetes requires a safe yet effectivetreatment. While synthetic drugs have been the primary approach for managing diabetes, their limitations warrant exploration of alternative solutions. As the prevalence of diabetes is spirally incrising, research into alternative treatments and potential sources of antidiabetic compounds becomes increasingly crucial. In recent years, the potential of medicinal plants, including orchids, has gained significant attention due to their unique bioactive compounds. With 44 orchid species falling under 21 genera traditionally used for diabetes treatment, validated by experimental studies conducted on 27 genera encompassing 56 species, orchids represent a particularly promising avenue for discovering natural and sustainable alternatives to conventional therapies. Bioactive compounds such as gigantol, moscatilin, lusianthridin, and syringic acid were frequently associated with antihyperglycemic effects. Mechanisms included antioxidant activity, enzyme inhibition (α-amylase and α-glucosidase), insulin sensitisation, β-cell regeneration, and modulation of key signalling pathways (e.g., PI3K/Akt, AMPK). Several species also exhibited protective effects against diabetes-induced complications.Orchids have the potential to revolutionise diabetes treatment by offering natural, effective, and sustainable alternatives to conventional drugs. However, further research including and clinical trials is warranted for effective understanding of the therapeutic efficacy of orchids and translate them into practical treatments. With continued exploration and investment in this field, anti-diabetic orchids may become an integral part of our pharmaceutical arsenal against diabetes.

Keywords: *Orchids; diabetes mellitus; traditional medicine; anti-diabetic;bioactive compounds.*

1. Introduction

Diabetes mellitus is one of the leading and prominentmetabolic disorder with a myriadof aetiologies that include chronic hyperglycaemia and abnormality in the carbohydrate metabolism by the human body, besides of carbohydrates, fats etc. due to lower blood insulin level or insensitivity of targeted organs to insulin [1]. In today's fast-paced and sedentary world, diabetes has become a significant health concern, impacting individuals of all ages and socioeconomic backgrounds. Moreover, it poses significant challenges to healthcare systems and individuals as they strive to manage and control the disease[2]. As the prevalence of diabetes continues to rise, research into alternative treatments and potential sources of antidiabetic compounds becomes increasingly crucial.

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In recent years, the potential of medicinal plants, including orchids, has gained significant attention due to their unique bioactive compounds.

Facts and figures reported in the latest 2021 edition of Diabetes Atlas released by the International DiabetesFederation have shown that the current global diabetic population of 536.6 million will be augmented to a staggering number of 783.2 million by 2045[3].

2. Classifications of Diabetes Mellitus

Diabetes mellitus is a multifaceted, chronic metabolic condition that poses a significant global health challenge due to its rising incidence and associated severe complications. Broadly, the disease is categorized into three distinct types.

Type 1 diabetes

Type I diabetes, historically known as insulin-dependent diabetes, arises from a severe deficiency in insulin production. This is primarily caused by the loss of functional pancreatic beta cells, often resulting from an autoimmune response where the body's immune system erroneously targets insulin-producing cells [4]. Consequently, individuals with this condition rely entirely on exogenous insulin administration to regulate blood glucose. Clinical onset typically occurs during childhood or early adolescence. While the precise etiology of this autoimmune disorder remains elusive, factors such as genetic predisposition, family history, dietary habits, sedentary behavior, and chronic pancreatitis are believed to precipitate its development [5].

Mechanistically, Type 1 diabetes is characterized by the immune-mediated destruction of pancreatic beta-cells by macrophages, as well as CD4+ and CD8+ T cells.

Islet cell antibodies—specifically those targeting glutamic acid decarboxylase (GAD) within β-cells—are detected in approximately 85% of patients. This destruction leads to insulin deficiency and metabolic dysregulation, further compounded by the dysfunction of pancreatic α-cells, which secrete excessive glucagon. Furthermore, unchecked lipolysis results in elevated free fatty acids, while reduced expression of hepatic glucokinase and GLUT-4 in adipose tissue impairs insulin sensitivity in target organs [6].

Type 2 diabetes

Type II diabetes (insulin-independent) is the most prevalent form of the disease, accounting for approximately 90% of the diabetic population. In this scenario, patients either fail to produce sufficient insulin, or their bodies are unable to utilize it effectively to maintain glycemic control. The condition is often exacerbated by comorbidities such as obesity, hypertension, hyperlipidemia, and cardiovascular disease [7]. T2DM is strongly linked to sedentary lifestyles, which contribute to chronic systemic inflammation characterized by proinflammatory markers including IL-6, CRP, TNF-α, and IL-1. Notably, IL-1 plays a critical role in the autoimmune aspect of the disease by activating the NF-kappaB transcription factor, which suppresses β-cell function and induces apoptosis [8]. Consequently, management strategies primarily focus on dietary modifications, physical activity, and pharmacotherapy [9].

Although traditionally an adult-onset condition, T2DM is characterized physically by a combination of insulin resistance and impaired insulin secretion due to β-cell degradation [10]. At the cellular level, Mitochondria-associated membranes (MAMs), responsible for lipid exchange and calcium signaling, contain key insulin signaling proteins; their dysfunction is a significant contributor to peripheral insulin resistance [6]. Additionally, microRNAs (miRNAs), which regulate various cellular processes, have been implicated in the pathogenesis of T2DM and are being investigated as potential biomarkers [11].

Gestational diabetes

The third category, gestational diabetes, is specific to pregnancy. It manifests as hyperglycemia in expectant mothers and affects approximately 7–10% of pregnancies globally [12].

3. Chronic Complications Associated with Diabetes

Diabetes mellitus is associated with a range of long-term physiological sequelae, including neuropathy, nephropathy, retinopathy, dyslipidemia, and various cardiovascular disorders [13]. These complications significantly diminish the quality of life and increase mortality rates among diabetic populations.

Cardiovascular and macrovascular pathologies: cardiovascular disease (CVD) remains a primary cause of morbidity in diabetic patients. Persistent hyperglycemia causes extensive damage to the vascular endothelium, accelerating the development of atherosclerosis. This process leads to the accumulation of arterial plaque, resulting in narrowed and hardened vessels that impede blood flow and heighten the risk of myocardial infarction and stroke. The management of comorbid factors—specifically obesity, hypertension, and dyslipidemia—is essential for mitigating cardiovascular risks in these patients [14].

Neurological impairment (diabetic neuropathy): Prolonged exposure to elevated blood glucose levels often results in diabetic neuropathy, characterized by widespread nerve damage. While this can affect various systems, it most commonly manifests in the distal extremities. Patients frequently experience a loss of tactile sensation, which increases the risk of undetected injuries, potentially leading to foot ulceration and, in severe instances, lower-limb amputation. Current research emphasizes the role of glycemic variability, suggesting that fluctuations in blood sugar levels are as detrimental as sustained hyperglycemia in the progression of nerve damage [15].

Renal dysfunction (diabetic nephropathy): Diabetic nephropathy is a critical complication involving the degradation of the kidney's filtration units. Over time, high glucose levels impair the kidneys' ability to process waste and excess fluids, which can progress to end-stage renal disease (ESRD) requiring dialysis or transplantation. Recent molecular studies have highlighted the influence of microRNAs (miRNAs) in this process; these non-coding RNAs appear to regulate the intercellular pathways responsible for renal pathogenesis, marking them as potential therapeutic targets for future interventions [16].

Ocular complications Diabetes also poses a severe threat to visual health, leading to conditions such as cataracts, glaucoma, and, most notably, diabetic retinopathy. The latter is driven by damage to the retinal vasculature and is characterized by increased vascular permeability, tissue ischemia, and pathological angiogenesis. Research has identified Vascular Endothelial Growth Factor (VEGF) as a primary mediator in the advancement of retinopathy [17]. If left unmanaged, these conditions can lead to permanent vision loss.

Immunological vulnerability and mental health: Beyond organ-specific damage, diabetes compromises the immune system, rendering patients more susceptible to bacterial and fungal skin infections, as well as recurrent urinary tract and yeast infections [18].

Furthermore, the disease imposes a substantial psychosocial burden. The rigorous demands of constant glucose monitoring, strict dietary adherence, and medication schedules can lead to chronic stress, anxiety, and clinical depression. This emotional strain is often compounded by the social stigma and the persistent fear of future physical complications [19].

Collectively, these complications underscore the necessity of early diagnosis, preventative strategies, and integrated management. Ongoing research into the molecular underpinnings of these pathologies is vital for curbing the global progression of diabetes and its associated burdens.

4. Drawbacks ofsynthetic drugs in diabetes treatment

Current medications environ an extensive variety of conventional synthetic drugs such as biguanides, α-glucosidase inhibitors(AGIs), thiazolidinediones, sulfonylureas and non-sulfonylureas secretagogues. The commonly used biguanide is metformin, which reduces hepatic glucose generation in the presence of insulin.α-glucosidase is an enzyme that breaks downcomplex carbohydrates into simpler forms. By inhibiting them, the AGIs slow down the uptake of carbohydrates and thus reduce postprandial blood sugar levels. Thiazolidinediones increase the uptake of glucose in the muscles and adipose tissues.

Orally administered class of drugs,sulfonylureas and non-sulfonylureas secretagogues bind to receptors in the beta cells, allowing the closure of potassium adenosine triphosphate (K_{ATP}) channels and entry of calcium into the cell, which in turn releases insulin[20].

Regardless of their effectiveness,synthetic drugs come with several drawbacks apart from limitations like cost effectiveness, ease of availability and toxicity. Firstly, they often have side effects on prolonged usage that can range from mild discomfort to severe complicationssuch as osteoporosis, obesity, hypoglycaemia, lactic acidosis, peripheral edema and abdominal discomfort [13]. Additionally, the continued use of synthetic drugs may lead to drug resistance, requiring high doses or alternate medications for treatment. Moreover, these drugs only manage the symptoms of diabetes and do not address the underlying cause of the disease. Therefore, the search for newer natural herbal drugs which are easily available, cost-effective, biologically safe and which do not require laborious pharmaceutical processes is desired as an alternative [21].

5. Botanical Sources as Alternative Therapeutics for Diabetes Mellitus

Since antiquity, botanical sources have provided a foundational basis for therapeutics, with numerous traditional medicinal systems highlighting the potent antidiabetic properties of various plant species. Ethnomedical practices have leveraged these natural properties for centuries to regulate and manage glucose levels [22]. Such usage is extensively documented across diverse global cultures, most notably in India [23, 24]. Historically, the Indian subcontinent has a rich legacy of utilizing indigenous botanical remedies for diabetes, with records dating back to the 6th century BC in the foundational texts of Charaka and Sushruta [25].

Phytochemical Composition and Mechanisms of Action The antidiabetic efficacy of medicinal plants is derived from a complex array of bioactive secondary metabolites, including terpenoids, saponins, flavonoids, carotenoids, alkaloids, and glycosides [26]. These phytoconstituents operate through multiple physiological pathways:

- Flavonoids: These compounds have been shown to facilitate cellular glucose uptake, stimulate insulin secretion, retard glucose absorption within the gastrointestinal tract, and inhibit key enzymes responsible for endogenous glucose production.
- Alkaloids: These molecules demonstrate significant hypoglycemic effects by enhancing insulin sensitivity and optimizing glucose metabolic pathways.

Beyond their biochemical efficacy, medicinal plants offer strategic advantages over synthetic pharmacological agents. They are often more accessible and cost-effective, particularly in regions where conventional pharmaceutical infrastructure is limited [27].Pharmacological and ethnobotanical scope extensive research, ranging from ethnobotanical surveys to rigorous pharmacological evaluations, continues to identify promising compounds for diabetes management. It is estimated that between 800 and 1,200 plant species possess inherent antidiabetic potential. A significant number of these have already demonstrated measurable bioactivity when subjected to modern experimental validation [28].

6. Orchids and Diabetes: A Comprehensive Overview

The Orchidaceae family is among the most expansive and diverse groups of flowering plants, comprising an estimated 25,000 to 35,000 species [29]. While widely celebrated for their ornamental and aesthetic appeal, orchids have increasingly become a focal point for pharmacological research due to their unique ecological adaptations and specialized secondary metabolites [30]. Beyond their horticultural value, members of the Orchidaceae family are recognized for significant antidiabetic properties [31]. This vast genetic diversity provides a rich reservoir for the discovery of novel bioactive compounds. Historical records indicate that Chinese traditional medicine was the first to document the therapeutic utility of orchids, many of which are now the subjects of rigorous phytochemical and pharmacological investigation [32].

India serves as a primary biodiversity hotspot for orchids, hosting approximately 2,500 species across 167 genera. Despite this abundance, the full therapeutic potential of Indian orchids remains underutilized due to a lack of comprehensive scientific validation and a limited understanding of their molecular mechanisms. There is an urgent need for standardized clinical research, the development of robust bioassays, and thorough toxicological evaluations using various animal models to ensure safety and efficacy [9]. Although the application of orchids in modern diabetes treatment is in its early stages, preliminary studies have demonstrated significant hypoglycemic activity, suggesting a promising role for these plants in managing hyperglycemia.

6.1 Bioactive constituents and molecular mechanisms

Orchids synthesize a diverse array of secondary metabolites that underpin their medicinal efficacy. These include phenols, flavonoids, alkaloids, tannins, steroids, phenanthrenes, stilbenoids, and various glycosides [33]. Many of these compounds have exhibited potent antidiabetic effects in both *in vitro* and *in vivo* models [34, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51]. Some of the key molecules are listed here: Alkaloids: Molecules such as dendrobine, erianin, and gigantol have shown efficacy in lowering blood glucose and mitigating diabetic cataracts; Stilbenoids: Thunalbene (3,3′-dihydroxy-5-methoxystilbene), first isolated from *Thunia alba*, possesses antioxidant, anti-inflammatory, and antidiabetic properties [33]; Gigantol: Found in *Dendrobium* species, it exhibits a broad pharmacological profile, including neuroprotective, vasorelaxant, and anti-cataractogenic activities [52]; Kinsenoside: Extracted from *Anoectochilus roxburghii*, it facilitates the restoration of pancreatic β-cells and regulates antioxidant enzymes to combat oxidative stress [34].

A primary mechanism for these effects is the reduction of oxidative stress and insulin resistance [53]. Chronic hyperglycemia triggers the overproduction of reactive oxygen species (ROS), which damages beta-cells and exacerbates insulin resistance. Furthermore, elevated glucose leads to the formation of Advanced Glycation End-products (AGEs). Antioxidant compounds found in various orchid species neutralize these free radicals, thereby improving insulin sensitivity and protecting cellular integrity [36][38][39][40][54][55][56][57][58][59][60][61][62][63][64][65].

6.2 Conservation status

The surge in demand for medicinal orchids has led to unsustainable harvesting and significant depletion of wild

populations. Habitat loss due to deforestation and urbanization further threatens these species. Consequently, many orchids are listed on the IUCN Red List of threatened species, and the entire Orchidaceae family is protected under Appendix II of CITES to regulate international trade [66].

6.3 Ethnomedicinal and pharmacological perspectives

A systematic literature review was conducted using databases such as Google Scholar, Science Direct, and PubMed Central to evaluate the antidiabetic potential of orchids. Ethnobotanical data reveals that 44 to 50 orchid species across 21 genera are traditionally employed to treat diabetes (Table 1). The genus *Dendrobium* is the most well-represented, with species like *D. officinale* being a staple in nearly 190 polyherbal formulations in China [74, 75]. Other notable genera include *Anoectochilus*, *Eulophia*, and *Dactylorhiza*, used across India, Thailand, Turkey, and Africa [28][59][79][80].

6.3.1 Pharmacological mechanisms of action

Pharmacological evidence (summarized in Table 2) identifies several key pathways through which orchids exert antidiabetic effects:

I. Digestive enzyme inhibition: Species such as *Dactylorhiza hatagirea* and *Dendrobium polyanthum* inhibit α-amylase and alpha-glucosidase, slowing carbohydrate digestion and reducing postprandial glucose spikes [32][79][94][95].

II. Antioxidant defense: Phenolics and polysaccharides from *Acampe praemorsa* and *Gastrodia elata* scavenge free radicals (DPPH, hydroxyl), preventing lipid peroxidation and protecting vital tissues [46][49][104].

III. Insulin signaling and islet protection: Extracts from *D. candidum* and *D. officinale* stimulate insulin secretion, promote beta-cell regeneration, and enhance insulin signaling via the AKT phosphorylation pathway [34][106][111].

IV. Glucose uptake and GLUT4 modulation: Orchids like *Anoectochilus burmannicus* and *D. loddigesii* upregulate GLUT4 expression, facilitating the transport of glucose into adipose and muscle tissues [62][113][114].

V. Anti-inflammatory activity: Orchid-derived compounds attenuate low-grade systemic inflammation by modulating cytokines like TNFα and CRP, thereby reducing insulin resistance [53][62][115].

VI. Hypolipidemic and hepatoprotective effects: Species like *D. nobile* regulate lipid metabolism by downregulating lipogenesis genes (e.g., *Srebp1*), reducing triglycerides, and improving hepatic function [106][108][116].

VII. Anti-AGE activity: *D. brymerianum* and *Prosthechea michuacana* prevent the glycation of proteins, which is critical in avoiding long-term vascular and neural complications [60][96][118].

VIII. Targeted organ protection:Nephroprotective: *D. officinale* preserves glomerular structure and improves renal biomarkers [107]; Retinoprotective: *D. chrysotoxum* and erianin inhibit VEGF expression to maintain the retinal barrier [43][48]; Cardioprotective: Polysaccharides mitigate diabetic cardiomyopathy by reducing oxidative stress [75];

Gut microbiota modulation: Recent evidence suggests *D. aphyllum* enhances beneficial microbial diversity and short-chain fatty acid (SCFA) production, aiding glucose metabolism [110]; Anti-obesity effects: Species like *D. delacourii* inhibit adipogenesis and reduce lipid accumulation, addressing a core risk factor for Type 2 Diabetes [53][97][120].

7. Constraints in the Development of Orchid-Based Antidiabetic Agents

Despite their therapeutic promise, the transition of orchids from traditional remedies to mainstream pharmaceutical agents faces several significant bottlenecks [121]. These challenges can be categorized into logistical, ecological, and regulatory domains.

I. Logistical and cultivation barriers: The commercial-scale production of orchids for drug development presents immense logistical hurdles. Orchid cultivation is notoriously labor-intensive and time-consuming, often requiring precise environmental control over temperature, humidity, and light—factors that make artificial propagation both complex and expensive [122, 123]. Furthermore, many species with high antidiabetic potential are endemic to remote, biodiversity-rich regions. Geographic barriers, political instability, and underdeveloped infrastructure in these areas often impede researchers' ability to secure sufficient samples for comprehensive study.

II. Ecological and conservation concerns: The extraction of bioactive compounds from wild orchids poses a direct threat to natural ecosystems. Orchids are highly sensitive to habitat loss caused by deforestation, climate change, and illicit harvesting [124]. With many species already classified as endangered, conservation is a critical priority. Responsible utilization necessitates the development of sustainable harvesting protocols and a commitment to preserving natural habitats [125].

III. Scientific and regulatory hurdles: The pathway to clinical approval is demanding. There is currently a significant disparity between folkloric reports and rigorous scientific validation. Specific data regarding the isolation, safety, and efficacy of individual bioactive compounds remain sporadic, and human clinical trials are virtually non-existent [28]. Additionally, the complex regulatory landscape—encompassing ethical considerations and the requirements for standardized clinical application—often deters pharmaceutical investment, thereby slowing the commercialization of orchid-derived treatments.

To bridge these gaps, several strategies are recommended: I. Biotechnological alternatives: Leveraging tissue culture and synthetic biology can facilitate the production of bioactive compounds without depleting wild populations [123]; II. Conservation advocacy: Implementing education campaigns and establishing protected areas can ensure the survival of valuable species while supporting research [107] and III. Streamlined regulation: Enhancing collaboration between researchers and regulatory bodies could optimize the approval process for plant-based therapies without compromising safety standards.

Table 1: Details of ethnomedicinal orchids used for the treatment and management of diabetes in different parts of the world

Name of orchid	Part used	Country	References
Acampe praemorsa (Roxb.) Blatt & McCann	Whole plant decoction	India	[85]
Agrostophyllum callosum Rchb.f.	Tuber, chewed	India	[86]
Anoectochillus burmannicus Rolfe.	Whole plant	China	[62]
*Anoectochilus formosanus*Hayata	Decoction of whole plant	China	[73][[76]
Anoectochilus roxburghii (Wall.) Lindl.	Decoction of whole plant	China	[77]
Arundinagraminifolia(D. Don) Hochr.	Decoction of root	India	[87]
Cremastraappendiculata (D.Don) Makino	Bulbs	China	[126][127]
Cyrtorchis arcuate (Lindl.) Schltr.	Whole plant grinded to powder	Africa	[28]
Dactylorhizahatagiria(D. Don) Soo.	Whole plant	India	[79]
Dactylorhizaromana(Sebast.) Soo.	Tubers	Turkey	[80]
Dendrobium aphyllum (Roxb.) C.E.C. Fisch.	Whole plant	Bangladesh	[67]
*Dendrobium aurantiacum*F. Muell.	Lead decoction infused	Australia	[68]
*Dendrobium aquem*Lindl.	Whole plant	India	[58]
Dendrobium candidum Wall ex Lindl.	Leaves decoction	India, China	[16][87]
*Dendrobium crepidatum*Lindl. et Paxton.	Whole plant	China	[32][46]
Dendrobium chrysotoxum Lindl.	Multiple use of the entire plant	China	[44][69]
*Dendrobium draconis*Rchb.f.	Stem	Thailand	[36]
Dendrobium formula (Herbadendrobii)	Herbal formula using whole plant	China	[28]
Dendrobium fimbriatum Hook.	Whole plant	China	[70]
*Dendrobium formosum*Roxb. Ex. Lindl.	Whole plant	Thailand	[45]
Dendrobium huoshanense C.Z. Tang et S.J Cheng	Stem	China	[71][72]
Dendrobium loddigesii Rolfe.	Whole plant	China	[64]
Dendrobium moniliforne(Linnaeus) Swartz	Whole plant	China	[57]
Dendrobium nobile Lindl.	Whole plant	China	[73]
Dendrobium officinale Kimura et Migo	Polyherbal formulation using whole plant	China	[74][75]
Eria tomentosa (J. Koenig) Hook.f.	Whole plant	Bangladesh	[67]
Eulophia epidendraea(Retz) Fischer	Tuber	India	[78]
*Eulophia herbacea*Lindl.	Tubers	China	[38]
*Eulophia ochreata*Lindl.	Tubers	India	[59]
Geodorumdensiflorum(Lam.) Schltr.	Whole plant/pseudobulbs	Bangladesh	[67]
*Grammatophyllumspeciosum*Blume	Whole plant	Thailand	[93]
*Malaxisrheedi*B. Heyne ex Wallace	Whole plant	India	[88]
Maxillariatenufolia Lindl.	Flower	China	[50]
Nerviliaplicata(Andrews) Schltr.	Whole plant decoction	India	[89]
*Orchis anatolica*Boiss.	Root	Jordan	[129]
Orchis latifolia Linn.	Root	India	[90]
Papilionanthe teres (Roxb.) Schltr.	Whole plant consumed	Bangladesh	[67]
Prosthechea michuacana (Lex.) W.E.Higgins	Bulbs	Mexico	[37][60]
Prosthechea karwinskii(Mart.) J.M.H. Shaw	Leaves are chewed and infusion of pseudobulbs and leaf decoction	America and Mexico	[92]
Spiranthes acaulis (Sm.) Cogn.	Whole plant is used in the initial stage of diabetes	Latin America, Caribbean	[81]
Spiranthes australis (R.Br.) Lindl.	Decoction of whole plant	China, Trinidad, Tobago	[68]
Spiranthes sinensis (Pers.) Ames.	Whole plants decoction	China, India	[28][82][83][84]
Rhychostylis retusa (L) Blume	Roots	Indonesia	[91]
Vanda tessellate(Roxb.) Hook. ex G.Don	Whole plant	India	[130]

Table 2: Pharmacological evidence of anti-diabetic potential of orchids

Sl. No.	Orchid Species	Model of Investigation	Major Findings	Isolated Bioactive Compounds	Inferences	References
1	*Acampepraemossa* (Roxb.) Blatt. & McCann.	1. Animal model: Albino Wistar rats	Hydroalcoholic extract showed dose dependent free radical activity.	Alkaloids, carbohydrates, phenols flavonoids, glycosides, sterols, terpenoids, tannins.	Antioxidant and bioactive compounds	[65]
		2. *In vitro* assay: Superoxide, DPPH, hydrogen peroxide	Aqueous and ethanol extracts showed dose dependent scavenging of radicals.	As abpve	Antioxidant and bioactive compounds	[99]
2.	*Anacamptis pyramidalis* (L.) Rich	1. *In vitro*phytochemical quantification	Antioxidant activity of methanol extract in various assaysindicating potential source of antioxidant	Disaccharide, parishinC,dihydroxybenzoic acid derivative, citric acid, parishin G, roseoside, caffeic acid derivative, acacetin derivative, gastrodin derivative, parishin B	Aantioxidant and anti-diabetic potential	[94]
		2. *In vitro* α-glucosidase and *α*-amylase enzyme inhibition	α-amylase and α-glucosidase inhibition by methanol extract) important for type 2 diabetes management.			
3	*Anoectochillus burmannicus* Rolfe.	*In vitro* cell line model: mature 3T3-L1 adipocytes	*A. burmannicus*extracts treated TNF-α adipocytes improved glucose uptake; ABE non-toxic by *in vitro* cytotoxic and *in vivo* assays	Chlorogenic acid, Coumaric acid, Ferulic acid, Vanillic acid	Anti-inflammation, anti-insulin resistance due to macrophage and adipocyte inflammation	[62]

4	*Anoectochilus formosanus*Hayata	1. Animal model: Streptozotocin induced diabetic model	Lowered fasting blood glucose, triglycerides, and total cholesterol, lower renal lipid peroxidation, increasing glutathione, catalase activity restoration	-	Anti-hyperlipidaemic and antioxidant	[54]
		2. *In vitro* FL83B cell line model	TNF-α induced cellsexhibited change in anti-insulin resistance and glucose uptake and anti-insulin resistance was improved by hot water	-	Anti-insulin resistant potential	[28]
5	*Anoectochilus roxburghii* (Wall.) Lindl.	1. Animal model: Alloxan induced hyperglycaemic mice	Water extract decreased blood glucose level and improved enzymatic antioxidant activity and vitamin E levels in kidney	-	Anti-diabetic and antioxidant	[56]
		2. Animal model: Streptozotocin induced diabetes	n-Butanol extract with kinsenoside reduced blood glucose level and protected β-beta cell of pancreatic islet	Kinsenoside	Anti-diabetic and antioxidant	[34]
		3. Clinical trial	Herb capsules supported recovery of diabetic-Type-2 patients	-	Anti-diabetic potential	[28]
		4. Animal model: Diabetic rats	Higher dose of n-Butanol extract increased the insulin leveland ß cell regeneration	Alkaloidsflavonoids, glycosides, organic acids, steroids, triterpenes etc.	Hypoglycaemic effect	[77]
		5. Animal model: Streptozotocin induced high fat diabetic rats	Improved antioxidant activity of the rats and enhanced the breakdown of glucose and lipids significantly	*Anoectochilus roxburghii* polysaccharosefrom the whole plant	Anti-diabetic activity and antioxidant activity	[100]
		6. Animal model: Streptozotocin induced high fat diabetic rats	ARP from different parts of *A. roxburghii*exhibited higher most antidiabetic activity	ARPs-1 and 2 from roots and leaves	Anti-diabetic activity	[101]
		7. Animal model: Streptozotocin induced high fat diabetic rats	The basic structure of ARP exhibited antidiabetic activities on streptozotocin induced diabetic rats is 1,3-ß-D-glucan	ARPs-p	Anti-diabetic activities anti-oxidant, anti-hyperlipidemic and anti-hyperglycemic activities	[105]
		8. Animal model: high fat diet fed rats	ARPs and exercise helps in reducing lipid accumulation and oxidative stress and controlling obesity	*Anoectochilusroxburghii* polysaccharide (ARP)	Anti-diabetic potential	[103]
6	*Aerides multiflora Wall.* ex J. Lindl.	1. *In vitro:* Biochemical analysis 2. *In vitro*: *α*-glucosidase enzyme inhibition	Isolated compounds tested for non-competitiveα-glucosidase inhibitory activity and Aerimultin C (compound 3) was found to be most potent.	Aerimultin, imbricatin, agrostonin, dihydroconiferyl dihydro-p- coumarate, 5-methoxy-9,10-dihydrophenanthre ne-2,3,7-triol,dihydrosinapyl dihydroferulate, 6-methoxy coelonin, gigantol	Anti-diabetic potential	[95]

7	*Arundinagraminifolia*(D.Don) Hochr.	1. *In vitro*: Biochemical analysis	Reported the presence phenanthrenes which maybe the cause of antioxidant activity	Phenanthrenes, essential oils, quinones, Stilbenoids, ketones, phenolic acidsglycosides	Antioxidant activity	[49]
		2. *In vitro:* Biochemical analysis	Six antioxidants potential phenanthrene secondary metabolites isolated	Five dihydrophenanthrenes and one diphenanthrene	Antioxidant activity	[50]
		3. *In vitro:* Biochemical analysis	Nine new glucosyloxybenzyl 2R-benzlymalate and two new glucosyloxybenzyl 2R-isobutylmalatederivatives isolated	Arundinoside L: 1,4 bis-(β-D-lucopyranosyloxybenzyl)-2-(β-D-glucopyranosyl-2-acetyl-6- > 1–2R-benzylmalyl)-2R-benzylmalic acid. Arundinoside M: 1,4 bis-(ß-D-lucopyranosyloxybenzyl) -2-(β-D-glucopyranosyl-2,4-diacetyl-6- > 1–2R-benzylmalyl)-2R-benzylmalic acid. Arundinoside O: 1-(ß-D-glucopyranosyloxybenzyl)-2-(ß-D-glucopyranosyl-2,3-biacetyl)-4-(ß-D-glucopyranosyloxybenzyl-6-acetyl)-2R-benzylmalate. Arundinoside P: 1-(β-D-glucopyranosyloxybenzyl)-2-(β-D-glucopyranosyl-2,3-diacetyl)-4-(β-D-glucopyranosyloxybenzyl-6-hydroxybenzyl)-2R-benzylmalate.	Antioxidant activity	[51]
8	*Bletilla striata* (Thunb.) Rchb. f. (BS)	Animal model: high fat diet (HFD)-fed mice	Improved glucose intolerance, insulin resistance and inflammation	Polysaccharides	Antidiabetic potential	[131]
9	*Calanthe fimbriata* Franch.	Animal model: Streptozotocin induced diabetic model and oral glucose tolerance test (OGTT) mice	Methanol root extract of with potent antihyperglycemic effects in OGTT and STZ-induced diabetic mice, improving liver function and glycogen content, but no effect on normal blood glucose levels in normoglycemic mice	Over 40 constituents characterized including organic acids, ester, and sterols, etc.	Antidiabetic potential	[117]
10	*Dactylorhiza hatagirea* (D.Don) Soo.	1. *In vitro*: α-glucosidase and α-amylase enzyme inhibition	Leave extract inhibited α-Amylase and α-Glucosidase enzymes	Albumin, butanedioic acid, hydroquinone, lesoglossin, militarrin, pyranoside, pyrocatechol, saponins, ascorbic acid, phylloquinones, naphthoquinones, glucomannan, carotenoids, and glycosidic compounds	Antidiabetic potential	[79]
		2. *In vitro*: 3T3-L1 cell line	Leave extract showed increased cellular uptake of glucose via GLUT4glucose transporter.	Qquercetin, carbohydrate and saponins	Antidiabetic potential	[114]
		3. *In vitro*: α-amylase enzyme inhibition	Root extract exhibited α-amylase inhibition.			
		4. Animal model: alloxanmonohydrate induced in wistar albino rats	Root hydroalcoholic extract helped in reducing blood sugar level, total cholesterol, and total triglycerides and improved total protein content			
11	*Dactylorhiza romana* (Sebast.) Soo.	*In vitro*: α-amylase and α-glucosidase	Tuber methanolic extract exhibited highest α-amylase activity against chloroform extract.	Gallic acid, chlorogenic acid, caffeic acid, catechin, protocatechuic acid, p-hydroxybenzoic acid, epicatechin, syringic acid, vanillin, p- coumaric acid, kaempferol, routine, rosmarinic acid, cinnamic acid, quercetin, and luteolin	Antioxidant and antimicrobial agent	[80]

12	*Dendrobium aqueum*Lindl.	*In vitro*: Biochemical analysis	Dose dependent increased free radical scavenging potential and to protect thiol-groups during glycation.	-	Antioxidant potential and anti-glycation potential	[58]
13	*Dendrobium aphyllum* (Roxb.) C.E.C. Fisch.	1. Animal model: Kunming mice model	Decreases blood glucose level, increased G6Pase and GDH enzymatic activity and up regulating the expression of glucose transporters GLUT1 and GLUT2	*Dendrobium aphyllum* polysaccharide (DAP)	Anti-diabetic potential	[110]
		2. *In vitro*: Biochemical analysis	New phenanthrene, aphyllone A (1) and four new bibenzyl derivatives, aphyllone B (2) and aphyllals C-D (3-5), together with nine known compounds (6-14) having DPPH radical scavenging activity	Aphyllone A (1) Aphyllone B (2) Aphyllal C (3) Aphyllal D (4) Aphyllal E (5)	Antioxidant activity	[42]
14	*Dendrobium aurantiacum*var. *denneanum*Kerr.	1. Animal model: Wistar rats	Gigantol compound isolated delayed lens turbidity and kept lens transparent through reduction in gene expression.	Gigantol	Alleviation of diabetic cataract.	[41]
		2. Animal model: Alloxan induced diabetic rat model	*D. denneanum*polysaccharide was able to reduce blood glucose level and increase glucose tolerance in alloxan-induced hyperglycaemia rats	*D. denneanum* polysaccharide	Hypoglycaemic activity	[132]
15	*Dendrobium brymerianum* Rchb.f.	*In vitro*: α-glucosidase enzyme inhibition	Plant extract showed inhibition of α- glucosidase and inhibition of AGEs formation	Flavonoid and phenols	Strong anti-diabetic potential and AGEs inhibitory activity	[96]
16	*Dendrobium candidum* Wall ex Lindl.	Animal model: Streptozotocin induced diabetic model	Elevated serum insulin levels and lowered serum glucagon, stimulation in beta cells and down stimulated alpha cells; Increased liver glycogen concentration in adrenaline-induced hyperglycaemic mice.	-	Anti-hyperglycaemic potential, insulin stimulatory effect; glycogen inhibitory effect	[128]
		2. Animal model: Streptozotocin induced diabetic model	Protective effects of the kidney by managing the vascular endothelial growth factor (VEGF), Glucose tranporter 1 (GLUT-1) and CTGF expressions.	-	Ameliorate diabetic nephropathy	[112]
		3. *In vitro:* Biochemical analysis	8 new bibenzyl derivatives isolated named dendrocandins J-Q (1-8) with potent antioxidant activity.	Dendrocandins J-Q (1-8)	Antioxidant activity	[39]
		4. *In vitro* cell line study: Human corneal epithelial cells (HCEC)	Improved HCEC cells growth in high-glucose condition and lowered apoptosis through regulation of Bax and Bcl-2 gene expression. Protected and repaired corneal epithelial cell damage caused by high glucose	Polysaccharides	Plays a crucial role in protecting and repairing corneal epithelial cell damage caused by high glucose levels.	[133]
17	*Dendrobium christyanum* Rchb. f	*In vitro*: α-glucosidase enzyme inhibition and glucose uptake stimulatory activities	Compounds 4 and 6 inhibits α-glucosidase and stimulating glucose uptake.	Methyl haematommate (1), methyl 2,4 dihydroxy-3,6-dimethylbenzoate (3), ndocosyl 4 hydroxy-trans- cinnamate (4), vanillin (5), coniferyl aldehyde (6), 4,5-dihydroxy-2-methoxy-9,10 dihydrophenanthre ne (7), gigantol (10), and diorcinolic acid (13)	Potential hypoglycemic agents	[134]

18	*Dendrobium chrysotoxum* Lindl.	1. Animal model: Alloxan induced hyperglycaemic mice	Dose-dependent scavenging potential polysaccharide isolated protected Jurkat cells from glucose oxidase mediated cytotoxicity	*Dendrobium chrysotoxum* Lindl polysaccharide	Antioxidation and hypoglycaemic activity.	[55]
		2. Animal model: Streptozotocin induced diabetic model	Reduced inflammation of retina in diabetic retinopathy by lowering the breakdown of retinal barriers and improving the occluding and claudin -1 expressions	-	Ameliorating diabetic retinopathy	[69]
		3. Animal model: Streptozotocin induced diabetic rats	Erianin, a naturally occurring compound blocked high glucose induced vascular endothelial growth factor expression in oxygen induced retinopathy and streptozotocin induced diabetic rats	Erianin	Ameliorating diabetic retinopathy	[43]
		4. *In vitro* cell line model: RF/6A cells & microglia BV-2 cells	Erianin controls high glucose induced retinal angiogenesis by blocking high glucose induced vascular endothelial growth factor expression through ERK1/2-mediated hypoxia-inducible factor 1-alpha (HIF-1α) transcriptional activation.	Erianin	Ameliorating diabetic retinopathy	[43]
		5. Animal model: Streptozotocin induced diabetic rats	Extract regulated the mRNA expression of vascular endothelial growth factor and VEGF receptor 2 responsible for the genesis and development of diabetic retinopathy.	-	Ameliorating diabetic retinopathy	[112]
		6. *In vitro:* Biochemical analysis	Gigantol isolated had inhibitory action on aldose reductase and AR gene expression	Gigantol	Diabetic anti-cataract potential	[44]
		7. Animal model: Streptozotocin induced C57BL/6 male mice; Primary human retinal endothelial cells (HRECs) and ARPE19 cell line	Erianin lessened the blood-retinal barrier (BRB) breakdown and improved both inner and outer BRB damage in human retinal and APRE19 cells.	Erianin	Alleviation of diabetic retinopathy	[48]
18	*Dendrobium crepidatum*Lindl. et Paxton.	1. *In vitro*: Biochemical analysis	Secondary metabolites from stem extract exhibited dose dependent free radical scavenging activities	Tetracosane, triacontane, stigmasterol, and some phenol derivatives (2-methoxy-4-vinylphenol, 2-methoxy-5-(1-propenyl)-phenol, p-mesyloxyphenol, and 2,6-dimethoxy-4-(2-propenyl)- phenol)	Antioxidant potential	[46]
		2. *In vitro*: Biochemical analysis	Indolizidine alkaloid isocrepidamine isolated showed significant hypoglycemic effect.		Antidiabetic potential	[47]
19	*Dendrobium delacouriiGuillaumin.*	1. *In vitro*: α-glucosidase enzyme inhibition	EtOAc extract showed potent inhibition of α-glucosidase	11 compounds were isolated. Phoyunnanin E, phoyunnanin C, densifloral B	Potential for management of diabetes and obesity.	[97]
		2. Mouse embryonic preadipocyte 3T3-L1 cell	EtOAc extract showed anti-adipogenic effect (49% inhibition at 5 μg/mL).			
20	*Dendrobium draconis*Rchb.f.	*In vitro*: Biochemical analysis	Isolation of a new compound namely 5-methoxy-7-hydroxy-9,10-dihydro-1,4-phenanthrenequinone showed significant antioxidant activity.	5-methoxy-7-hydroxy-9,10-dihydro-1,4-phenanthrenequinone	Antioxidant activity	[36]
21	*Dendrobium ellipsophyllum*Tang & Wang.	*In vitro*: α-glucosidase enzyme inhibition	Whole plant extract showed inhibition of α- glucosidase	Flavonoid and phenols	Strong anti-diabetic potential	[96]
22	*Dendrobium fimbriatum* Hook.	Animal model: Sprague-Dawley rats	β cell apoptosis inhibition and pancreatic inflammation. Decreased hepatic lipid accumulation, lipid transport and oxidoreductase activity	-	Antidiabetic potential	[120]

23	*Dendrobium formosum*Roxb. ex. Lindl.	*In vitro* Cell line model: L6-myoblast cells of rats	Isolation of 12 bioactive molecules. Compounds 1 and 12 showed higher -glucosidase inhibitory activity. Lusianthridin (6) and moscatilin (11) had higheractivity than insulin. Glucose sensitivity improved by Lucianthridin.	Confusarin (1), hircinol (2), erianthridin (3), gigantol (4), nudol (5), lusianthridin (6), coelonin (7), dihydroconiferyl dihydro-p- coumarate (8),batatasin III (9), 2,5,7-trihydroxy-4-methoxy-9,10-dihydrophenanthre ne (10), moscatilin (11), and 5- methoxy-7-hydroxy-9,10- dihydro-1,4-phenanthrenequin one (12)	Antidiabetic and anti-obesity compound	[45]
24	*Dendrobium gibsonii*Paxton	*In vitro*: α-glucosidase enzyme inhibition	α-glucosidase inhibitory activityas a non-competitive inhibitor ofα-glucosidase	Dihydrodengibsinin, dendrogibsol, ephemeranthol A, dengibsinin, nobilone, aloifol I, lusianthridin, denchrysan A and 4-methoxy-9H-fluorene-2,5,9-triol	Anti-diabetic potential	[135]
25	*Dendrobium huoshanense* C.Z. Tang et S.J Cheng	1. Animal model: Streptozotocin induced diabetic cataract in animals	Blood sugar level decreased, and opacity of lens lessened in treated animals.	Polysaccharides	Diabetic anti-cataract potential	[71]
		2. Animal model: Streptozotocin induced diabetic model	Lowered fasting blood glucose, glycosylated serum protein and serum insulin but the glucose tolerance and the insulin sensitivity elevated	Polysaccharide GXG	Antidiabetic potential	[136]
		3. Animal model: Alloxan induced Male Kunming diabetic mice	Extract from frozen dried stems of *Dendrobium huoshanense*, *D. Officinale*, *D.nobile*and *D. Chrysotoxum*induced lower oxidative stress level; serum insulin level and fasting glucose level improved	Polysaccharides	Antidiabetic and antioxidant potential	[61]
26	*Dendrobium loddigesii* Rolfe.	1. *In vitro*: Biochemical analysis	Isolation of phenanthrenes showed significant inhibitory activity against nitric oxide production	LoddigesiinolsA(1) and loddigesiinols B(7) and stibenesloddigesiinols D (9).	Antioxidant activity	[35]
		2. *In vitro*: α-glucosidase inhibition	Isolation of new polyphenols responsible for lowering blood glucose	Loddigesiinols G-J.	Strong inhibition ofα-glucosidase observed	[40]
		3. Animal model: diabetic db/db mouse	Lowered inflammation, oxidative stress and blood glucose	Phenols	Antidiabetic and antioxidant potential	[64]
		4. 3T3-L1 cell line study	Lowered body weight, blood sugar, and serum lipid levels. The expression of peroxisome proliferator-activated receptor α (PPARα) and glucose transporter 4 (GLUT4) in liver were enhanced.	Shihunine, a water-soluble Orchidaceae alkaloid	Reduces lipid component and shows anti-diabetic activity.	[113]
27	*Dendrobium moniliforne* (Linnaeus) Swartz	1. Animal model: Male C57BL/6 mice	Lowered serum glucose, total cholesterol concentration and renal lipid accumulation	-	Anti-obesity effect and reno-protective effect.	[57]
		2. *In vitro*: LLC-PK1 renal epithelial cell line	Prevented kidney cell damage induced by oxidative stress	-	Antioxidant activity	[57]

28	*Dendrobium nobile* Lindl.	1. Animal model: Adrenalin and high-fat diet induced diabetes	Fatty acid β-oxidation genes Acox1 and Cpt1a, lipolysis gene ATGL and antioxidant gene metallothionein-1 and NADPH quinone oxidoreductase-1 (Nqo1) in livers of mice were upregulated while lipid synthesis regulator Srebp1 (sterol regulatory element-binding protein-1) was down regulated.	Alkaloids	Enhances glucose and lipid metabolism in liver hence helps in diabetic related metabolic disorders.	[137]
		2. Animal model: C57BL/6 mice	Enhanced hepatic lipid homeostasis, decreased cholesterol absorption and increased cholesterol excretion.	Alkaloids	Improved hepatic lipid homeostasis	[116]
29	*Dendrobium officinale* Kimura et Migo	1. Animal model: Alloxan induced diabetic animals	Fasting blood glucose level, glycosylated serum protein level is considerably reduced while serum insulin level is increased.	-	Hypoglycaemic activity	[28]
		2. Animal model: Alloxan induced hyperglycaemic mice	Oxidative stress level declined; serum insulin and fasting glucose level improved; reduced oxidative stress in kidney and liver	Polysaccharide	Antidiabetic and antioxidant activity	[61]
		3. Animal model: Streptozotocin and Adrenalin induced hyperglycaemia	Crude extract increased pancreas β cells and decreased α cells activities	-	Hypoglycaemic activity *in vivo*	[28]
		4. Animal model: Antidiabetic rats	Down-regulated the phosphorylation of JNK at Thr-183/Tyr- 185 residues and upregulated the phosphorylation of AKT at Ser-473 residue in the Islet tissues of Pancreas.	Polysaccharides, phenanthrenes, bibenzyls, saccharides and glycosides, essential oils, alkaloids	Hypoglycaemic activity *in vivo*	[106]
		5. Animal model: Streptozotocin induced diabetic model	Water extract showed an increase in liver glycogen and decrease in blood sugar. Stimulated an increase in energy and amino acid metabolism.	*O*-acetylglucomannan	Antidiabetic potential	[109]
		6. Animal model: Streptozotocin induced diabetic model	Reduced toll-like receptors, inflammatory response and urinary glucose.	-	Ameliorating diabetic cardiomyopathy	[75]
		7. Animal model: Streptozotocin induced diabetic model	Reduces oxidative stress, inflammatory cytokines and cardiac lipid accumulation.	Polysaccharides	Anti-diabetic activity	[108]
		8. Animal model: Streptozotocin induced diabetic model	Stem extract protected degeneration of hepatic glycogen and hepatic gluconeogenesis, repressed serum glycogen level, regenerated islets cells in pancreas.	Polysaccharides	Alleviation of diabetic complications	[103]
		9. Animal model: Streptozotocin induced type 1 diabetic model	Prevented oxidative stress.	Mannose, ribose, rhamnose, glucuronic acid, galacturonic acid, glucose, galactose and xylose-glucomannan	Alleviation of diabetic nephropathy	[107]
		10. Animal model: Streptozotocin induced diabetic model	Regulated the cells of glomerulus to normalcy, improved urea cycle, lipid, glucose, amino acids. metabolism	Glucomannan	Alleviation of diabetic nephropathy	[119]

29	*Dendrobium officinale* Kimura et Migo	11. H9c2 cardiomyocytes	Improved survival rate, reduced lipid peroxidation damage, endogenous antioxidant enzymes, inhibited the production ROS, lowered the mitochondrial membrane potential, down-regulated pro-apoptosis protein and up-regulated anti-apoptosis protein. Ameliorated H_2O_2-induced oxidative injury.	Polysaccharide (DOP-GY)	Ameliorating diabetic cardiomyopathy	[34]
		12. RAW 264.7 macrophages	Prevented cell death, improve oxidative lesions, increase cell viability.	Polysaccharides, DOPA-1 and DOPA-2 mainly comprised d-mannose and d-glucose	Antioxidant potential	[138]
		13. *In vivo:*Male C57BL mice	Aqueous extract activated the glucose metabolism related proteins in a concentration dependent manner.	-	Antidiabetic potential	[139]
		14. *In vitro:* C_2C_{12} cell line			Antidiabetic activity by activating insulin signalling pathway	
		15. *In vitro* LPS acting THP-1 cells	Protects THP-1 cells from LPS-induced cytotoxicity by inhibiting ROS and suppressing toll-like receptor-4expression	Polysaccharides	Antidiabetic potential	[115]
30	*Dendrobium. Parishii*Rchb.f.	*In vitro*: α-amylase inhibition	Whole plant extract showed inhibition of α-amylase with IC_{50} 46.57µg/ml.	Flavonoid and phenol	Strong antidiabetic potential	[96]
31	*Dendrobium polyanthum* Wall. ex Lindl.	*In vitro*: α-amylase and α-glucosidase inhibiting activity and advanced glycation end products formation	Compound 5 as a potent non-competitive inhibitor of alpha-glucosidase and compound 2 for its strong anti-AGE activity	Tectoquinone, moscatilin, ephemeranthol A, gigantol, moscatin, lusianthridin, 2,4,7-trihydroxy-9,10-dihydrophenanthrene	Potential of specific compounds with promising antidiabetic mechanisms.	[118]
32	*Dendrobium scabrilingue*Lindl.	*In vitro*: α-glucosidase enzyme inhibition	Whole plant extract showed inhibition of Advanced glycation end products formation	Flavonoid and phenol	Strong antidiabetic potential and AGEs inhibitory activity	[96]
33	*Dendrobium venustrum*Teijsm. & Binn.	*In vitro*: Biochemical analysis	Lusianthridin reduces lipid peroxidation, lowered formation of oxidized lipid products, preserves important lipid levels, suggesting its potential as a therapeutic agent to anti-atherosclerosis in thalassemia patients	Lusianthridin	Inhibited hemin-induced oxidative stress in low-density lipoprotein, demonstrating protective effects against LDL oxidation.	[95]
34	*Diaphananthebidens*(Afzel. Ex Sw) Schltr	Animal model: Streptozotocin induced diabetic rat	Methanolic leaf extract reduces blood glucose	Saponins, steroids, tannins and terpernoids	Anti-diabetic potential	[140]
35	*Eria graminifolia*Lindl.	*In vitro*: α-amylase inhibition	Pseudobulb extract showed dose dependent inhibition of α-amylase	Alkaloid, flavonoids, saponin, tannin	Potent source for antidiabetic phytochemicals	[98]
36	*Eulophia epidendraea* (Retz)Fischer	Animal model: Alloxan monohydrate induced	Lowered glucose level but increase in body weight of diabetic rats. Reduced kidney and liver weights	-	Hypoglycemic activity	[78]
37	*Eulophia herbacea* Lindl.	Animal model: Alloxan-induced hyperlipidaemic and diabetic rats	Glucomannan was isolated responsible for lowering in blood glucose level. Lowered triglycerides, total cholesterol and low-density lipoproteins and increase in high density lipoproteins.	Carbohydrates, proteins, amino acids, flavonoids, tannins, phenolics, mucilage, steroids, vitamins, and glucomannan	Antioxidant, hypoglycemic and hypolipidemic effects	[38]
38	*Eulophia ochreata* Lindl.	1. *In vitro*: Biochemical analysis	Antiglycation potential, alpha- amylase inhibitory activity and antioxidant.	Phenolic and flavonoids compounds	Anti-glycation and antioxidant	[59]
		2. *In vitro:* α-amylase enzyme inhibition				
39	*Gastrodia elata* Blume (GE)	Human umbilical vein endothelial cells (HUVECs)	Improved oxidative stress and inflammatory conditions in endothelial cells induced by high glucose levels.	Phenolic compounds (gastrodin, p-hydroxybenzyl alcohol (HBA), p-hydroxybenzaldehyde (HBZ))	Antidiabetic potential and antioxidant activity.	[104]

40	*Gastrochilusdistichus*(Lindl.) Kuntze	*In vitro*: α-amylase inhibition	Whole plant extract exhibited dose dependent inhibition of α-amylase	Flavonoids, steroids, saponin, terpenoids, tannin	Potent source for antidiabetic phytochemicals	[98]
41	*Grammatophyllumspeciosum*Blume	*In vitro*: α-glucosidase inhibiting activity	Ethanolic extract from 6-year-old rhizome+root (fresh and dry samples) showed strong activity	-	α-glucosidase inhibitor activity suggesting potential source of natural antidiabetic medicine	[93]
42	*Gymnadenia orchidis* Lindl	Animal model: Streptozotocin induced adult female albino mice	Inhibits hemoglobin glycation, normalizes the lipid profile, enhances antioxidant status, reduces lipid peroxidation	Terpenoids	Potential to counter diabetes associated oxidative stress and cellular damage	[141]
43	*Malaxisrheedii*B. Heyne ex Wallace	*In vitro*: α-amylase inhibitory and *α*-glucosidase inhibitory activity	*α*-amylase inhibitory activity and *α*-glucosidase inhibitory activity	Flavonoid, tannin, glycoside, resin,steroids, terpenoids, cardiac glycosides and triterpenoids etc.	Antidiabetic potential	[88]
44	*Maxillaria tenuifolia* Lindl.	*In vitro*: *α*-Glucosidase inhibitory activity	EtOAc extract exhibited inhibition of α-glucosidaseand supresses the carbohydratesdegradation.	3,4-dihydroxy benzoic acid methyl ester (1), flavanthridin (2), vanillic acid (3) and mangiferin (4)	Anti-hyperglycaemic and antioxidant activity	[50]
45	*Nerviliaplicata*(Andrews) Schltr.	Animal model: Streptozotocin induced diabetic rat model	Reduced blood sugar glucose, serum urea and creatinine levels. Restored damage in kidney tissue and reduced lipid peroxidation in kidneys.	-	Antidiabetic and regenerative potential	[89]
46	*Orchis anatolica*Boiss.	Animal model: Albino rats	Root extract lowered blood sugar levels, insulin releasing tendencies and reduction in cholesterol.	Flavonoids, triallate, theobromade, and tannins	Potent anti diabetic, anti-hyperglycaemic	[142][143]
47	*Orchis latifolia* Linn.	1. *In vitro*: α-amylase inhibition activity	Methanolic extract of roots showed significant inhibition of α-amylase activity	-	Anti-hyperglycaemic activity	[90]
		2. Animal model: Streptozotocin induced diabetic rats	Root methanolic extract lowered blood glucose and malondialdehyde levels and normalized anti-oxidative enzymes superoxide dismutase and catalase to normal levels.	-	Anti-hyperglycaemic, antioxidant and anti-lipid peroxidative activity	
48	*Otochilus albus* Lindl.	*In vitro*: α-amylase inhibition	Pseudobulb extract showed dose dependent inhibition of α-amylase.	Alkaloid, flavonoids, tannin	Antidiabetic potential phytochemicals	[98]
49	*Papilionantheuniflora*(Lindl.) Garay	*In vitro*: α-amylase inhibition	Whole plant extract showed dose dependent inhibition of α-amylase.	Alkaloid, flavonoids, steroids, terpenoids, tannin	Potent source for antidiabetic phytochemicals	[98]
50	*Pholidota articulata* Lindl.	*In vitro*: α-amylase inhibition	Pseudobulb and leaves extract exhibited dose dependent α-amylase inhibition	Alkaloid, flavonoids, steroids, terpenoids, tannin	Potent source for antidiabetic phytochemicals	[98]
51	*Prosthechea karwinskii* (Mart.) J.M.H. Shaw	1. Animal model: Metabolic syndrome induced Wistar rats	Leaves extract had antioxidant activity and reducing effect on glucose level, while flower extract lowered cholesterol and triglycerides	Flavonoid	Antidiabetic potential and antioxidant activity.	[63]
		2. Animal model: Metabolic syndrome induced Wistar rats	Oral administration of the extract controlledweight gain, decreased abdominal and pericardial fat deposits, and improved insulin resistance.	chlorogenic acid, Quinic acid, rutin,neochlorogenic, kaempferol-3-O-β-rutinoside, and embelin, TNF-α, HS-CRP, and adiponectin levels	Effective alternative for managing metabolic syndrome related conditions.	[53]

52	*Prosthechea michuacana* (Lex.) W.E.Higgins	1. Animal model: Streptozotocin induced diabetic Wistar albino rats	Crude extract of bulbs helped in lowering blood sugar, triglyceride, total cholesterol levels and hyperinsulinemia	-	Antidiabetic potential	[144]
		2. Animal model: Streptozotocin induced Type- 2 diabetic model	Two new tetracyclic triterpenoids from bulbs potent for insulin release, reduced blood glucose level, decreased total cholesterol and triglycerides	24-methyl, 24-hydroxy-5α-lanosta-9(11), 25-dien-3α-acetate and 24-methyl-24-hydroxy-5-lanosta 9 (11)-en-3α acetate	Hypoglycaemic and hypolipidemic potentials	[37]
		3. Both *In vitro* and *In vivo* streptozotocin-induced diabetic rats	Compounds preventedformation of advanced glycation end points and inhibited GHb and HbA1c. Exhibited antioxidant properties and protective effects against diabetic renal damage in streptozotocin-induced diabetic rats	α- α′-dihydro, 3′,5′,2-trimethoxy-3-hydroxy-4-acetyl-4′- isopentenyl stilbene, 4,6,7-trihydroxy-2-methoxy-8- (methylbut-2-enylphenan-thren-1-1′-4′,6′,7′- trihydroxy-2′-methoxy-8′-(methylbut-2′-enyl)-phenanthrene and gigantol	Strong anti-glycation agent and antioxidant	[60]
53	*Rhynchostylis retusa* (L) Blume.	*In vitro*: α-amylase inhibition, glucose diffusion method	Aqueous extract exhibited inhibition of glucose diffusion, anti-inflammatory, antioxidant properties	1-Methylene-2b-Hydroxymethyl-3,3-Dimethyl-4b-(3-ethylbut-2-Enyl)-Cyclohexane,2-Methylthiolane	Antidiabetic potential and antioxidant potential.	[65]
54	*Thunia alba* (Lindl.) Rchb.f.	*In vitro*: Biochemical analysis	Identified several phenolic compounds.	2,4,7-trihydroxy-9,10-dihydrophenanthrene; 2,8-dihydroxy-3,4,7-trimethoxyphenanthrene; 7-hydroxy-2,4-dimethoxy-9,10-dihydrophenanthrene (orchinol); 2,7-dihydroxy-1-(p-hydroxybenzyl)-4-methoxy-9,10-dihydroxyphenanthrene	Antidiabetic potential	[145]
55	*Vanda cristata* Wall. ex. Lindl.	*In vitro*: α-amylase inhibition	Stem extract showed dose dependent inhibition of α-amylase	Alkaloid, flavonoid,saponin, steroid, terpenoids and tannin	Potent source for antidiabetic phytochemicals	[98]
56	*Vanda tessellate*(Roxb.) Hook. ex G.Don	Animal model: Alloxan induced adult albino rats	Whole plant extract showed significant decrease in the blood sugar level.	-	Glibinclamide, likeantidiabetic standard drug.	[130]

8. Conclusion

The emerging interest in the antidiabetic properties of orchids represents a significant opportunity for pharmacological innovation. Intensified research into the bioactivity and molecular mechanisms of these plants is essential to unlock their full potential as targeted therapeutic agents.By integrating these natural compounds into modern medicine, we may reduce the current reliance on synthetic pharmaceuticals and move toward a more integrated, holistic approach to diabetes management. While the journey from "nature's pharmacy" to the clinical setting requires extensive validation through rigorous trials, the exploration of orchids underscores the vast, untapped potential of botanical resources. Continued investigation into this diverse family may yet yield critical breakthroughs for global health and well-being.

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