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Original Article

Screening of Bioactive Phytochemicals from *Gentiana kurroo* for Diabetes Management: An Integrated Molecular Docking and *in vitro* Enzyme Inhibition Study

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ABSTRACT

Background: Diabetes mellitus is a multifactorial metabolic disorder requiring multi-target therapeutic strategies. Medicinal plants rich in bioactive phytochemicals represent promising alternatives to conventional antidiabetic drugs. *Gentiana kurroo*, an endangered Himalayan herb, is traditionally used for metabolic and inflammatory disorders and contains compounds with potential glucose-lowering activity. **Objectives:** This study aimed to evaluate phytochemicals from *G. kurroo* for antidiabetic potential using molecular docking against key diabetic targets followed by *in vitro* enzyme inhibition assays. **Materials and Methods:** Five phytoconstituents (amarogentin, gentiopicroside, gentianine, gentianidine, and isogentisin) were docked against PPAR-γ (3DZY), glucokinase (4IXC), SIRT6 (3K35), GSK-3 (3F7Z), and tyrosine kinase (1IR3) using AutoDock Tools 1.5.6, with acarbose as reference. Drug-likeness and ADME profiles were predicted using SwissADME and pkCSM. Based on docking performance, gentiopicroside and isogentisin were further evaluated *in vitro* for α-amylase and α-glucosidase inhibition. **Results:** Gentiopicroside and isogentisin showed strong binding affinities (-7.2 to -9.0 kcal/mol) across multiple targets with stable hydrogen-bond and hydrophobic interactions. Both compounds complied with Lipinski's rule of five and exhibited favorable ADME profiles. *In vitro* assays demonstrated significant, concentration-dependent inhibition of α-amylase and α-glucosidase, comparable to acarbose ($p<0.05$). **Conclusion:** The combined *in silico* and *in vitro* findings suggest that gentiopicroside and isogentisin from *G. kurroo* are promising multi-target antidiabetic candidates. Further *in vivo* and clinical studies are required to validate their therapeutic potential.

Keywords: Diabetes, *Gentiana kurroo*, Gentiopicroside, Molecular docking, α-Amylase.

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INTRODUCTION

Diabetes Mellitus (DM) is a group of chronic metabolic disorders characterized by persistent hyperglycemia due to impaired insulin secretion, insulin action, or both. It has become a major global health concern, with its prevalence rising sharply in both developed and developing nations. In 2017, approximately 424.9 million people were affected worldwide. By 2019, this number had increased to over 463 million adults, and projections estimate it may reach 578 million by 2030 and 700 million by 2045 (Federation, 2019). The burden is significantly higher in urban areas (10.8%) compared to rural settings (7.2%). DM is broadly categorized into Type 1 diabetes, Type 2 diabetes, and Gestational Diabetes Mellitus (GDM). Type 1 diabetes is an autoimmune disorder resulting in the destruction of pancreatic β-cells, leading to insulin deficiency. Type 2 diabetes, the most prevalent form, is associated with insulin resistance and progressive β-cell dysfunction. GDM occurs during pregnancy, typically in the second or third trimester, affecting 2-10% of pregnancies, and is linked to hormonal changes that reduce insulin sensitivity. The pathogenesis of diabetes involves a complex network of interrelated biochemical pathways. Key mechanisms include oxidative stress, protein kinase C activation, hexosamine biosynthetic pathway flux, formation of Advanced Glycation End-products (AGEs), and activation of the polyol pathway. These overlapping mechanisms contribute not only to the onset of hyperglycemia but also to the development of long-term complications such as nephropathy, neuropathy, and retinopathy (Bailey and Day, 1989; DeFronzo, 1999). Current therapeutic agents target specific aspects of this complex network; however, they often exhibit limited efficacy in halting disease progression and are associated with adverse effects or drug resistance (DeFronzo, 1999). Consequently, there is an urgent need for novel, multi-target therapeutic approaches that can modulate various biochemical pathways involved in diabetes pathophysiology. Medicinal plants represent a promising source of such therapeutic agents. Numerous phytochemicals

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derived from traditional herbs have shown potential in regulating glucose metabolism and improving insulin sensitivity (Bailey and Day, 1989; He *et al*., 2017). Among these, *Gentiana kurroo*, an endangered medicinal herb native to the Himalayas, has garnered attention for its wide spectrum of pharmacological activities. Traditionally used for treating fever, inflammation, and liver disorders, *G. kurroo* contains several bioactive compounds such as amarogentin, gentiopicroside, gentianine, gentianidine, and isogentisin, many of which exhibit glucose-lowering and antioxidant properties (Manner and Humpf, 2014). Given the potential of these phytoconstituents to interact with multiple molecular targets, the present study aims to explore their efficacy using both *in silico* molecular docking and *in vitro* enzyme inhibition assays. The targets selected include PPAR-γ (3DZY), Glucokinase (4IXC), SIRT6 (3K35), GSK-3 (3F7Z), and Tyrosine kinase (1IR3), key regulators of glucose homeostasis, insulin signaling, and lipid metabolism. This dual approach seeks to identify natural compounds capable of modulating multiple diabetic targets simultaneously, thereby offering a holistic and effective strategy for diabetes management.

MATERIALS AND METHODS

In silico Study

Compound Selection and Preparation

In this molecular docking investigation, key phytoconstituents of *Gentiana kurroo* were selected based on literature evidence supporting their antidiabetic and antioxidant potential. Compounds such as amarogentin, gentiopicroside, gentianine, gentianidine, and isogentisin have been reported to act on various diabetes-related biochemical pathways, including oxidative stress, polyol pathway flux, protein kinase C activation, intracellular AGE formation, and the hexosamine biosynthetic pathway (Manner and Humpf, 2014; Bailey and Day, 1989; Oboh and Rocha, 2008). The 2D structures of the selected ligands were drawn using ChemDraw Ultra 7.0 and their corresponding 3D structures were generated and energy minimized. These molecular structures were then retrieved or cross-verified from the PubChem database. The ligand files were converted into PDBQT format after the addition of Gasteiger charges using AutoDock Tools (version 1.5.6).

Target Protein Preparation

Five diabetes-associated targets - PPAR-γ (3DZY), glucokinase (4IXC), SIRT6 (3K35), GSK-3 (3F7Z), and tyrosine kinase (1IR3) - were obtained from the RCSB Protein Data Bank. Proteins were prepared by removing water and native ligands, adding hydrogens and Kollman charges, energy minimization, and saving in PDBQT format using AutoDock Tools following Trott and Olson (2010) (Trott and Olson, 2012).

Molecular Docking Protocol

Docking simulations were performed using AutoDock Tools v1.5.6. Active sites were defined using optimized grid parameters generated by AutoGrid. Docking was executed with the Lamarckian Genetic Algorithm against five protein targets, using acarbose as reference. Binding energies and interaction details were analyzed from .dlg files (Trott and Olson, 2012).

Visualization and Interaction Analysis

Protein-ligand interactions were visualized using PyMOL (Pettersen *et al*., 2004), and further analyzed with the Protein-Ligand Interaction Profiler (PLIP) to characterize hydrogen bonds, π-π stacking, hydrophobic contacts, and salt bridges (Salentin *et al*., 2015). Docking results were ranked by binding energy and interaction stability to identify promising compounds for *in vitro* evaluation.

In vitro Anti-Diabetic Assays

α-Amylase and α-glucosidase inhibition assays were performed *in vitro* to evaluate the antidiabetic potential of amarogentin, gentiopicroside, gentianine, gentianidine, and isogentisin at different concentrations, using acarbose as the standard drug. Based on molecular docking results, Gentiopicroside and Isogentisin were selected for detailed enzyme inhibition studies targeting key carbohydrate-metabolizing enzymes. The assays were conducted in 96-well microplates following established protocols (Bernfeld, 1955; Kim *et al*., 2005). Briefly, 30 µL of test compounds (25-100 µg/mL) was incubated with 60 µL of enzyme solution (1 U/mL, PBS pH 6.9) at 37°C for 10 min. The reaction was terminated with 1 M HCl and 5% KI, and absorbance was measured at 630 nm. All experiments were performed in triplicate, and percentage inhibition was calculated using standard equations.

$$\%\ \text{of enzymes inhibition}\ (\%) = \frac{(\text{The absorbance of the control}\ )\ (\text{Absorbance of the sample})}{\text{The absorbance of the control}} \times 100$$

ADME and Drug-likeness Evaluation

Swiss ADME and pkCSM online tools were used for the prediction of Absorption, Distribution, Metabolism, Excretion, and Toxicity (ADMET) profiles (Diana *et al*., 2017; Pires *et al*., 2015). Drug-likeness was evaluated using Lipinski's Rule of Five.

Statistical Analysis

Statistical analysis was carried out using GraphPad Prism 6 software. Data are presented as mean±Standard Deviation (SD). Statistical significance was evaluated by one-way ANOVA followed by Dunnett's multiple comparison post hoc test (*p<0.05, **p<0.01, ***p<0.001). Duncan's multiple range test was also used where applicable (p<0.05).

RESULTS

Molecular Docking Analysis

Molecular docking was performed to evaluate the binding affinities of Amarogentin, Gentiopicroside, Gentianine, Gentianidine, and Isogentisin against five diabetes-related protein targets: PPAR-γ (3DZY), Glucokinase (4IXC), SIRT6 (3K35), GSK-3 (3F7Z), and Tyrosine kinase (1IR3). Binding energies ranged from -6.0 to -9.0 kcal/mol across all ligand-protein complexes. Isogentisin exhibited the strongest binding affinity toward Glucokinase (-9.0 kcal/mol) and showed consistently favorable binding energies across all targets (-7.9 to -9.0 kcal/mol). Gentiopicroside also demonstrated strong binding, particularly with Glucokinase (-8.9 kcal/mol), along with stable interactions with other targets. Key molecular interactions included hydrogen bonding, π-π stacking, and hydrophobic contacts. Isogentisin formed hydrogen bonds with THR65, CYS220, and TYR473, π-π stacking interactions with PHE282, and van der Waals interactions with LEU330. Gentiopicroside formed hydrogen bonds with HIS435, CYS220, and THR65, supported by van der Waals interactions with ALA292 and LEU330. Amarogentin, Gentianine, and Gentianidine exhibited moderate binding affinities stabilized by hydrogen bonds and hydrophobic interactions. Detailed binding energies and interaction profiles are presented in Table 1, while representative 2D and 3D interaction diagrams are shown in Figure 1.

In vitro Anti-diabetic Activity

Enzyme Inhibition by Selected Compounds at 100 µg/mL

At 100 µg/mL, Gentiopicroside and Isogentisin showed strong α-amylase (87.3%, 85.9%) and α-glucosidase (89.1%, 88.5%) inhibition, comparable to acarbose. Amarogentin showed moderate, and Gentianine and Gentianidine lower inhibition. IC_{50} values reflected these trends (Table 2, Figure 2).

Concentration-Dependent Enzyme Inhibition by Gentiopicroside and Isogentisin

Gentiopicroside and Isogentisin were evaluated for dose-dependent inhibition at 25-100 µg/mL, showing increasing α-amylase and α-glucosidase inhibition with concentration. Maximum inhibition occurred at 100 µg/mL, with lower activity at reduced doses (Table 3).

Table 3 showing the Concentration-Dependent Enzyme Inhibition by Gentiopicroside Isogentisin.

ADME Profiling

ADME and toxicity predictions using SwissADME and pkCSM indicated Amarogentin violated Lipinski's rules and had low gastrointestinal absorption. Gentiopicroside, Gentianine, Gentianidine, and Isogentisin complied with Lipinski's criteria and showed high absorption. No compounds were predicted hepatotoxic or mutagenic; BBB permeability was observed only for Gentianine and Gentianidine (Table 4).

DISCUSSION

The present study employed an integrated *in silico* and *in vitro* approach to comprehensively evaluate the antidiabetic potential of selected phytoconstituents. This multi-level strategy generated converging evidence on molecular interactions, functional enzyme inhibition, and pharmacokinetic suitability, thereby strengthening the biological relevance of the findings. Molecular docking analysis identified Isogentisin and Gentiopicroside as the most promising candidates, exhibiting strong and consistent binding affinities toward multiple diabetes-related protein targets,

Table 1: Showing Molecular Docking Results (Binding Affinity and Interaction.

Compound	PPAR-γ (3DZY)	Glucokinase (4IXC)	SIRT6 (3K35)	GSK-3 (3F7Z)	Tyrosine Kinase (1IR3)	Key Interactions
Amarogentin	-6.8 kcal/mol	-6.7 kcal/mol	-8.9 kcal/mol	-8.6 kcal/mol	-8.7 kcal/mol	H-bond: CYS220, HIS323; Hydrophobic: PHE282, LEU330
Gentiopicroside	-7.2 kcal/mol	-8.9 kcal/mol	-8.6 kcal/mol	-7.3 kcal/mol	-7.3 kcal/mol	H-bond: HIS435, CYS220, THR65; van der Waals: ALA292, LEU330
Gentianine	-6.4 kcal/mol	-7.1 kcal/mol	-6.1 kcal/mol	-6.4 kcal/mol	-6.0 kcal/mol	H-bond: THR65; Hydrophobic: VAL135, PHE282
Gentianidine	-6.0 kcal/mol	-7.0 kcal/mol	-6.1 kcal/mol	-6.5 kcal/mol	-6.0 kcal/mol	H-bond: SER342; van der Waals: VAL135, ALA292
Isogentisin	-7.5 kcal/mol	-9.0 kcal/mol	-7.9 kcal/mol	-8.1 kcal/mol	-8.0 kcal/mol	H-bond: THR65, CYS220, TYR473; π-π stacking with PHE282; van der Waals: LEU330

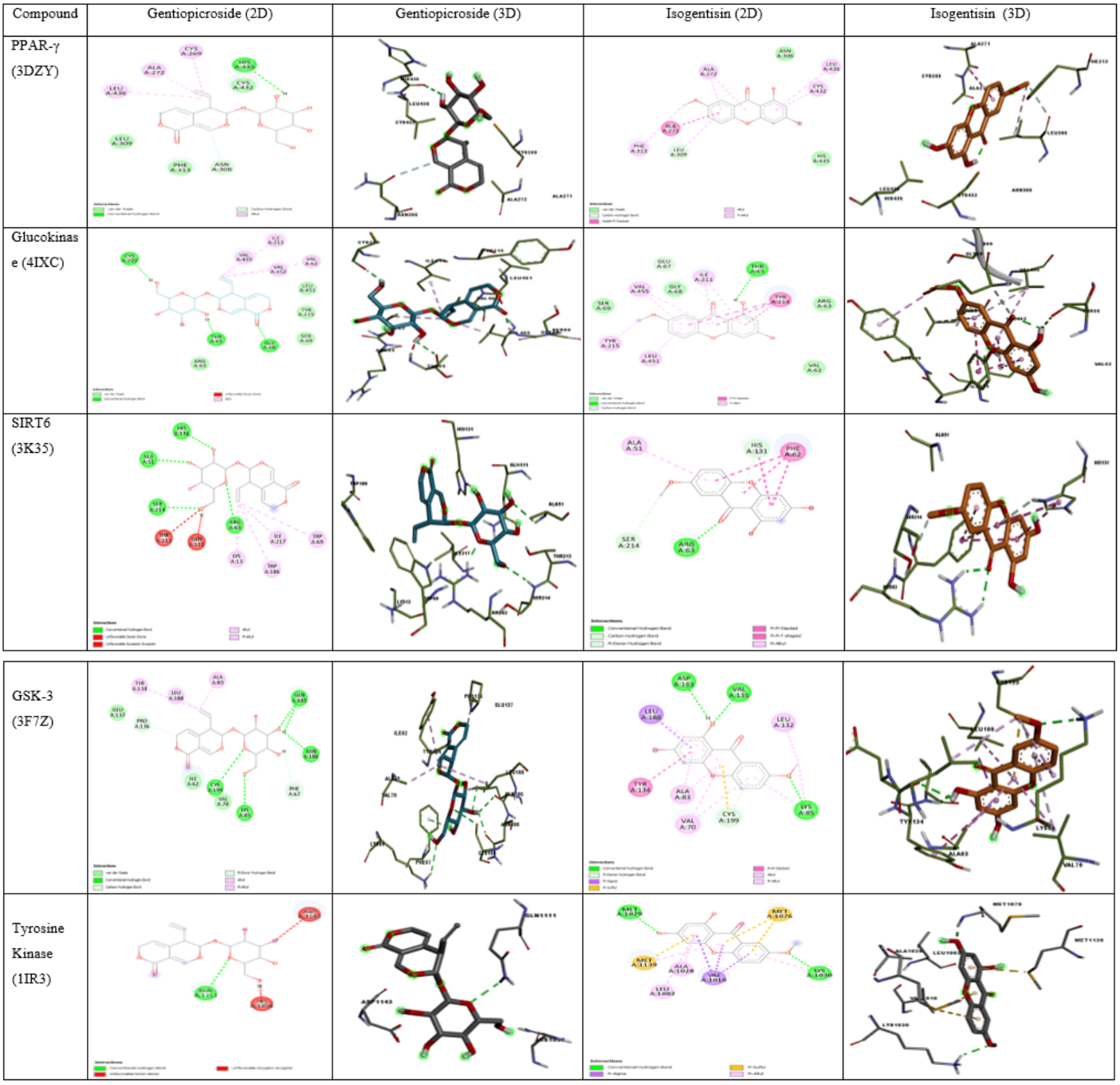

Figure 1: 2D and 3D structure of Gentiopicroside and Isogentisin on PPAR-γ (3DZY), Glucokinase (4IXC), SIRT6 (3K35), GSK-3 (3F7Z), Tyrosine Kinase (1IR3).

supporting a multitarget mechanism of action. Such multitarget activity is particularly advantageous in diabetes management, given the complex dysregulation of glucose metabolism, insulin signaling, and associated metabolic pathways. Among the evaluated targets, glucokinase plays a pivotal role in glucose sensing and insulin secretion, while PPAR-γ, GSK-3, SIRT6, and tyrosine kinase are critically involved in insulin sensitivity, glycogen synthesis, lipid metabolism, and intracellular signaling. The favorable binding energies observed for Isogentisin and Gentiopicroside across these targets suggest their potential to modulate interconnected pathways governing glycemic control. Increasing evidence indicates that natural compounds capable of interacting with multiple molecular targets may offer therapeutic advantages over single-target synthetic drugs, particularly in chronic metabolic disorders such as diabetes. The stability and specificity of ligand-protein complexes were supported by extensive hydrogen bonding, hydrophobic interactions, and π-π stacking. Hydrogen bonds with catalytically important residues, including THR65 and CYS220, contribute significantly to binding specificity and complex stability, while π-π stacking interactions

Table 2: Showing the Enzyme Inhibition by Selected Compounds at 100 µg/mL.

Compound	α-Amylase Inhibition (%) (Mean±SD)	α-Glucosidase Inhibition (%) (Mean±SD)	α-Amylase IC_{50} (µg/mL)	α-Glucosidase IC_{50} (µg/mL)
Acarbose	89.2±1.3	91.7±1.1	56.3	48.9
Amarogentin	73.5±2.2 ***	75.4±2.0 ***	71.4	68.2
Gentiopicroside	87.3±1.6 ns	89.1±1.3 ns	58.7	50.1
Gentianine	65.7±2.8 ***	68.2±2.5 ***	80.5	76.3
Gentianidine	61.4±3.1 ***	64.5±2.6 ***	85.1	78.9
Isogentisin	85.9±1.5 ns	88.5±1.2 ns	59.2	51.3

(Note: ns=not significant; $p<0.05$ = *, $p<0.01$ = **, $p<0.001$ = ***).

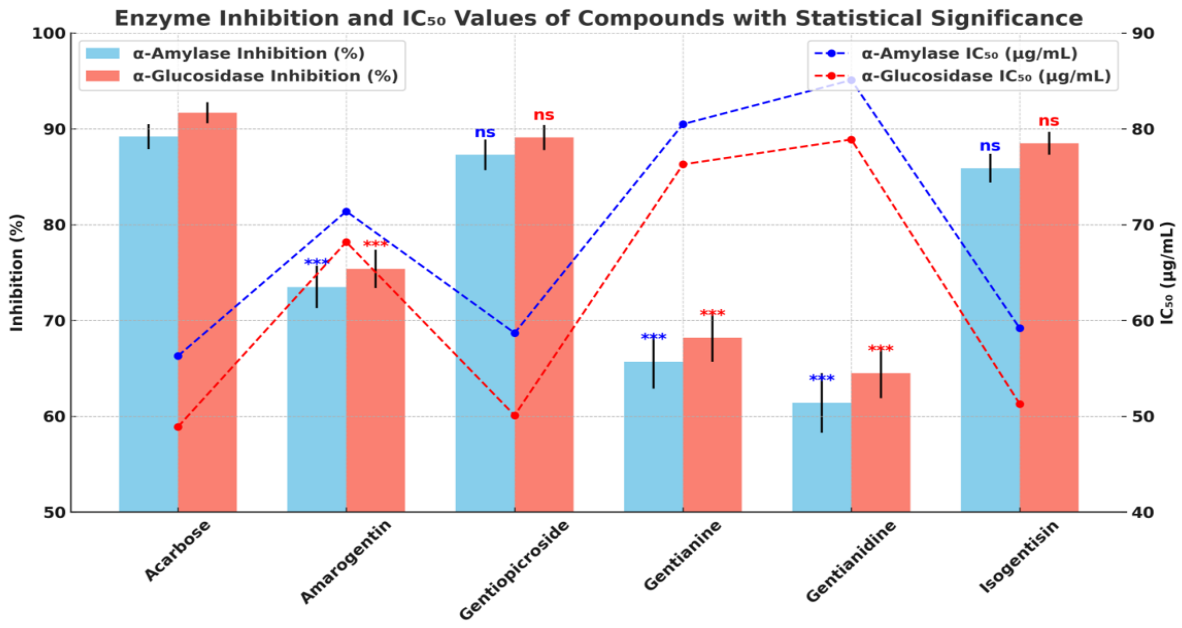

Figure 2: showing the enzyme inhibition and IC_{50} values of compounds with statistical significance.

enhance affinity by stabilizing aromatic moieties within enzyme active sites. These interaction patterns indicate effective molecular recognition and favorable ligand orientation. Comparable interaction profiles have been reported for established antidiabetic phytochemicals such as quercetin and epicatechin, which demonstrate multitarget binding and improve glycemic control through enzyme inhibition and insulin-sensitizing mechanisms (Ali *et al.*, 2024; Zu *et al.*, 2021). The consistency of these observations supports the mechanistic plausibility of the present findings. The *in vitro* enzyme inhibition assays further validated the molecular docking results. Gentiopicroside and Isogentisin exhibited potent inhibitory activity against both α-amylase and α-glucosidase, with inhibition percentages and IC_{50} values comparable to acarbose, a clinically established antidiabetic drug. Inhibition of these digestive enzymes delays carbohydrate digestion and reduces intestinal glucose absorption, thereby lowering postprandial hyperglycemia. The strong agreement between computational docking scores and experimental enzyme inhibition data reinforces the reliability of the *in silico* predictions and confirms the functional relevance of the observed molecular interactions. In contrast, Amarogentin exhibited moderate enzyme inhibition, while Gentianine and Gentianidine showed comparatively lower activity. These differences may be attributed to weaker binding affinities, fewer stabilizing interactions, or suboptimal molecular orientation within enzyme active sites. Such structure-activity relationships highlight the importance of specific functional groups and interaction patterns in determining biological efficacy. These findings are consistent with previous reports indicating that phenolic and flavonoid compounds with optimal hydrogen-bonding capacity and aromatic features tend to exhibit enhanced antidiabetic activity (Patel *et al.*, 2019). The concentration-dependent inhibition observed for Gentiopicroside and Isogentisin further supports their pharmacological relevance. A clear dose-response

Table 3: Concentration-Dependent Enzyme Inhibition by Gentiopicroside and Isogentisin.

Concentration (µg/mL)	% α-Amylase Inhibition (Mean±SD), Gentiopicroside	% α-Amylase Inhibition (Mean±SD), Isogentisin	% α-Glucosidase Inhibition (Mean±SD), Gentiopicroside	% α-Glucosidase Inhibition (Mean±SD), Isogentisin
100	87.3±1.6 (ns)	85.9±1.5 (ns)	87.3±1.6 (ns)	85.9±1.5 (ns)
75	70.8±1.4 (***)	72.3±1.3 (***)	70.8±1.4 (***)	72.3±1.3 (***)
50	53.6±1.1 (***)	55.8±1.0 (***)	53.6±1.1 (***)	55.8±1.0 (***)
25	32.7±0.8 (***)	34.1±0.7 (***)	32.7±0.8 (***)	34.1±0.7 (***)

Note: ns = not significant; $p<0.05$ = *, $p<0.01$ = **, $p<0.001$ = ***.

Table 4: Showing the ADME Profiling.

Property	Amarogentin	Gentiopicroside	Gentianine	Gentianidine	Isogentisin
Molecular Weight (g/mol)	524.47	402.37	223.24	237.28	298.27
H-bond Donors	6	5	1	1	2
H-bond Acceptors	13	9	3	4	5
LogP (Lipophilicity)	-0.42	-0.30	1.12	1.30	1.05
Gastrointestinal (GI) Absorption	Low	High	High	High	High
Lipinski's Rule of Five Violations	2 Violations	No Violation	No Violation	No Violation	No Violation
Blood-Brain Barrier (BBB) Permeability	No	No	Yes	Yes	No
Hepatotoxicity	No	No	No	No	No
AMES Toxicity (Mutagenicity)	No	No	No	No	No

relationship was evident, with increasing concentrations leading to enhanced inhibition of both α-amylase and α-glucosidase. Dose dependency is a desirable characteristic for therapeutic agents, as it reflects predictable biological responses and facilitates rational dose optimization. Similar concentration-dependent enzyme inhibition profiles have been reported for other natural antidiabetic compounds, reinforcing the translational potential of these findings (Anshika *et al*., 2022). ADME and drug-likeness analysis indicated that Gentiopicroside and Isogentisin comply with Lipinski's criteria, show good gastrointestinal absorption, and lack predicted hepatotoxicity or mutagenicity. In contrast, Amarogentin showed Lipinski violations and lower absorption. Limited blood-brain barrier permeability suggests reduced CNS-related adverse effects. Overall, the combined *in silico* and *in vitro* findings identify Gentiopicroside and Isogentisin as promising multitarget antidiabetic candidates with effective enzyme inhibition and favorable pharmacokinetic profiles. However, the absence of *in vivo* and clinical data warrants further pharmacodynamic, bioavailability, and long-term safety studies in appropriate animal models to support clinical translation.

CONCLUSION

The present study demonstrates the antidiabetic potential of selected phytoconstituents through an integrated *in silico* and *in vitro* approach. Molecular docking analysis identified Gentiopicroside and Isogentisin as the most promising compounds, exhibiting strong and consistent binding affinities toward multiple diabetes-related targets, including glucokinase, PPAR-γ, GSK-3, SIRT6, and tyrosine kinase. These interactions were supported by favorable hydrogen bonding, hydrophobic contacts, and π-π stacking interactions, suggesting stable ligand-protein complexes. *In vitro* enzyme inhibition assays further confirmed the computational findings, with both compounds showing potent α-amylase and α-glucosidase inhibitory activities comparable to acarbose, along with clear concentration-dependent responses. ADME and drug-likeness predictions indicated favorable pharmacokinetic and safety profiles, particularly for Gentiopicroside and Isogentisin. Overall, the results highlight these compounds as promising multitarget antidiabetic candidates. However, further *in vivo* studies and clinical investigations are required to validate their efficacy, bioavailability, and long-term safety before therapeutic application.

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ABBREVIATIONS

DM: Diabetes Mellitus; **PPAR-γ:** Peroxisome Proliferator-Activated Receptor Gamma; **GSK-3:** Glycogen Synthase Kinase-3; **SIRT6:** Sirtuin 6; **ADME:** Absorption, Distribution, Metabolism, and Excretion; **BBB:** Blood-Brain Barrier; **GI:** Gastrointestinal; **IC_{50}:** Half Maximal Inhibitory Concentration; **SD:** Standard Deviation; **ANOVA:** Analysis of Variance.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUTURE SCOPE AND LIMITATION

Future studies should emphasize *in vivo* validation of *Gentiana kurroo* phytomolecules in diabetic models. Evaluation of synergistic effects, formulation strategies to improve bioavailability, and comprehensive preclinical and clinical pharmacokinetic and toxicity assessments are essential.

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