

Exploring the Mechanism of *Ganoderma lucidum* Active Components against Diabetic Tubulopathy Based on Network Pharmacology and Molecular Docking

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Abstract [**Objectives**] This study was conducted to explore the mechanisms of active components from *Ganoderma lucidum* against diabetic tubulopathy based on network pharmacology and molecular docking. [**Methods**] Small-molecule compounds of *G. lucidum* were screened from the TCMSP database ($OB \geq 55\%$, $DL \geq 2$), and their targets were predicted via SwissTargetPrediction. Known molecular targets of *G. lucidum* polysaccharides (GLP) were obtained through literature review, and the two tracks were merged to obtain the drug target set. Disease-related targets of diabetic tubulopathy were retrieved from the GeneCards database (relevance score ≥ 30), and common targets were obtained by intersection with the drug targets. A PPI network was constructed using the STRING platform, and topological analysis was performed with Cytoscape software to screen key targets. GO and KEGG pathway enrichment analyses were conducted. Molecular docking was performed using AutoDock Vina to verify the binding affinity between key components and targets. [**Results**] A total of 17 active small molecules of *G. lucidum* were identified (4 steroids and 13 triterpenoids), yielding 168 unique targets. Literature mining of GLP provided 43 targets. The two tracks were merged, resulting in 200 drug targets. Intersection with 10 918 disease targets yielded 184 common targets. The PPI network comprised 182 nodes and 2 317 edges, and 47 hub genes were identified (degree ≥ 36). Among them, IL6, TNF, AKT1 and IL1B showed the highest degree values. hnRNPF exhibited only weak PPI connections with TNF, AKT1, and HIF1A (scores 0.154–0.186), suggesting its involvement in the disease network through post-transcriptional regulation. The AGE-RAGE signaling pathway was the most significantly enriched in the KEGG analysis (24 genes, $FDR = 2.18 \times 10^{-23}$). The results of molecular docking showed that Ganosporelactone B exhibited a binding energy of -10.22 kcal/mol with hnRNPF and of -13.27 kcal/mol with HIF1A, demonstrating a multi-target binding advantage. [**Conclusions**] The active components of *G. lucidum* exert anti-diabetic tubulopathy effects through multi-component, multi-target, and multi-pathway mechanisms, and the AGE-RAGE signaling pathway served as a central mechanistic hub. hnRNPF may be directly targeted by *G. lucidum* triterpenoids and participate in the disease process through post-transcriptional regulation.

Key words *Ganoderma lucidum*; Diabetic tubulopathy; hnRNPF; Network pharmacology; Molecular docking; AGE-RAGE signaling pathway

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As the primary driving factor of mortality and disability in diabetes, diabetic kidney disease (DKD) has become the leading cause of end-stage renal disease worldwide^[1–3]. For a long time, glomerulosclerosis has been regarded as the core pathological hallmark of DKD. However, recent research paradigms are shifting—tubulointerstitial lesions are not merely bystanders secondary to glomerular injury, but independently initiate in the early stages of DKD, with their degree of damage not parallel to that of glomerular lesions^[4–5]. Against this background, the concept of "diabetic tubulopathy" has expanded the research focus from the glomerulus to tubular epithelial cells, revealing the early dynamics of pathological processes such as oxidative stress, inflammatory cascades, and epithelial-mesenchymal transition under the hyperglycemic microenvironment.

Heterogeneous nuclear ribonucleoprotein F (hnRNPF) is an important member of the hnRNP family, involved in the regulation of alternative splicing through specific recognition of G-tract sequences in pre-mRNA^[6]. Recent studies have suggested that hnRNPF is associated with tubular cell apoptosis and podocyte

injury in diabetic tubulopathy^[7–8]. However, few reports have been conducted on whether it participates in the pathogenesis of diabetic tubulopathy and whether it could serve as a therapeutic target.

Ganoderma lucidum (Lingzhi) is a traditional and precious Chinese medicinal herb. Its pharmacologically active substances include small-molecule triterpenoids/steroids and macromolecular polysaccharides^[9–11]. *G. lucidum* polysaccharides (GLP) have been reported to possess hypoglycemic, insulin resistance-improving, and renoprotective activities^[12–14]. However, due to their macromolecular nature, polysaccharides cannot be analyzed by conventional TCMSP screening workflows, resulting in most existing network pharmacology studies on *G. lucidum* being limited to small-molecule components^[15–17]. In this study, a "dual-track strategy" was adopted to integrate small-molecule database screening with literature-based target mining and to thereby construct a "drug-target-pathway" network of active components from *G. lucidum* against diabetic tubulopathy, and molecular docking was further performed to verify key targets. This study provides a systematic computational interpretation of the molecular mechanism of *G. lucidum* against diabetic tubulopathy.

Materials and Methods

Screening of active components from *G. lucidum* and target prediction

Information on small-molecule components of *G. lucidum* was

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obtained from the TCMSP database (<https://tcmisp-e.com/>). To ensure the pharmacokinetic feasibility of the selected components, dual filtering criteria were set with oral bioavailability (OB) $\geq 55\%$ and drug-likeness (DL) ≥ 2 . After screening, a set of active compounds with favorable absorption and drug-like properties was obtained. Subsequently, the canonical SMILES structures of each compound were used for target prediction on the SwissTargetPrediction platform (<http://www.swisstargetprediction.ch/>). The prediction model was set to *Homo sapiens*, and all predicted targets with probability values greater than zero were retained. After standardization of gene names via UniProt and removal of duplicates, a small-molecule target set (hereafter referred to as "Track A") was established.

Literature-based target mining for GLP

Given that macromolecular polysaccharides cannot be subjected to target prediction through the TCMSP-SwissTargetPrediction workflow, a literature mining strategy was adopted to construct the GLP target set. Systematic literature retrieval was conducted in the PubMed and CNKI databases using the keywords "*G. lucidum* polysaccharide" in combination with "diabetic nephropathy", "kidney", "target", and "mechanism". Retrieved papers were read individually, and molecular targets of GLP validated through *in vivo* or *in vitro* experiments were extracted. After deduplication, the resulting target set was designated as "Track B," which was then merged with Track A to form a complete dual-track drug target set.

Collection of disease targets and identification of common targets

Disease-related targets were obtained from the GeneCards database (<https://www.genecards.org/>). Five keywords with progressive pathological hierarchy, namely "diabetic kidney disease", "diabetic nephropathy", "diabetic tubulopathy", "renal fibrosis" and "renal tubular injury", were used for independent retrieval separately. The results from each retrieval were filtered using a relevance score ≥ 30 as the cutoff to retain targets with high relevance. After merging the five sets and removing duplicates, a disease target set was established. The identification of common targets between drugs and diseases was performed using the Venny 2.1.0 online tool for intersection analysis.

PPI network construction

Protein-protein interaction data for the common targets were obtained from the STRING database (<https://string-db.org/>). The search parameters were set to the *H. sapiens* proteome, with a minimum required interaction score threshold of 0.4 (medium confidence). The interaction records were downloaded in TSV format and imported into Cytoscape 3.10.0 to construct a visual network. Three topological parameters, namely, degree, betweenness, and closeness centrality, were calculated for each node using the built-in Network Analyzer tool. The median degree value of the network was multiplied by 2, and the obtained value was used as the screening threshold for hub genes. To characterize the interaction features of hnRNPF, it was explicitly included in the network analysis. It should be noted that the STRING database erroneously mapped the interaction partners of VDR to CYP27B1

(which is actually a vitamin D-metabolizing enzyme). In this study, the correct interaction relationship of VDR was manually supplemented through literature verification.

GO enrichment and KEGG pathway analysis

Functional annotation and pathway enrichment were performed using the built-in enrichment analysis API of the STRING database. Functional annotation was conducted for two branches of the Gene Ontology (GO), biological process (BP) and molecular function (MF), as well as for the KEGG metabolic and signaling pathway database. Multiple testing correction was performed using the Benjamini-Hochberg method, with a corrected FDR < 0.05 as the significance inclusion criterion. Graphical representation of the enrichment results was generated using the Bioinformatics Online Platform (<http://www.bioinformatics.com.cn/>).

Molecular docking

Six pathway-representative target proteins were selected as receptors for molecular docking. Among them, hnRNPF and HIF1A lack experimentally determined crystal structures and were therefore modeled using AlphaFold-predicted structures (UniProt IDs: P52597 and Q16665, respectively). Crystal structures of AKT1 (PDB: 3O96), TNF (PDB: 1TNF), PPARG (PDB: 2P4Y), and MTOR (PDB: 4JSV) were obtained from the RCSB PDB database. All protein structures were preprocessed using OpenBabel prior to docking, including removal of water molecules, addition of polar hydrogens, and assignment of Kollman charges, followed by export in PDBQT format. On the ligand side, 10 representative compounds covering all chemical structure types (steroids, triterpenoid acids, triterpenoid aldehydes, triterpenoid ketones, triterpenoid alcohols, and triterpenoid lactones) present in this study were selected. Their 3D structures were preferentially obtained from the PubChem database; and compounds for which only 2D structures were available were converted to 3D conformations using RDKit via distance geometry-based conformational generation. Docking calculations were performed using the AutoDock Vina v1.2.7 engine. The docking box was centered at the geometric center of the active pocket of each protein, with side lengths set to 24 or 30 Å to cover binding regions of different sizes. The exhaustiveness parameter was set to 8. For each docking run, the conformation with the lowest binding energy among the output poses was taken as the representative conformation.

Results and Discussion

Screening of active components from *G. lucidum*

A total of 17 active compounds from *G. lucidum* were identified through screening of the TCMSP database (Table 1), including 4 steroids (cervisterol, ergostadienol, β -sitosterol, and brassicasterol) and 13 triterpenoids (comprising subclasses such as triterpenoid acids, triterpenoid aldehydes, triterpenoid ketones, triterpenoid alcohols, and triterpenoid lactones). The prediction via SwissTargetPrediction yielded 846 target records, which were reduced to 168 targets after deduplication (Track A).

Table 1 Information of active compounds from *G. lucidum*

Compound name	SMILES	PubChem CID	PubChem download URL
Cervisterol	<chem>C[C@H](/C=C/[C@H](C)C(C)C)[C@H]1CC[C@@H]2[C@@]1(CC[C@H]3C2=C[C@H]([C@@]4([C@@]3(CC[C@H](C4)O)C)O)O)C</chem>		
Ergostadienol	<chem>CC(C)C(C)C=CC(C)C1CCC2C1(CCC3C2=CCC4C3(CCC(C4)O)C)C</chem>	314387	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/314387/SDF?record_type=3d
β -Sitosterol	<chem>CC[C@H](CC[C@@H](C)[C@H]1CC[C@@H]2[C@@]1(CC[C@H]3[C@H]2CC=C4[C@@]3(CC[C@@H](C4)O)C)C(C)C</chem>	222284	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/222284/SDF?record_type=3d
Campestdienol	<chem>CC(C)C(C)C=CC(C)C1CCC2C1(CCC3C2=CCC4C3(CCC(C4)O)C)C</chem>	314387	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/314387/SDF?record_type=3d
Epoxyganoderiol A	<chem>CC(CCC1C(O1)(C)CO)C2CCC3(C2(CCC4=C3C(CCC5C4(CCC(=O)C5(C)C)C)O)C)C</chem>	14015434	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/14015434/SDF?record_type=3d
Epoxyganoderiol B	<chem>CC(CCC1C(O1)(C)CO)C2CCC3(C2(CC=C4C3=CCC5C4(CCC(=O)C5(C)C)C)C)C</chem>	14015436	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/14015436/SDF?record_type=3d
Ganoderic acid DM	<chem>C[C@H](CC/C=C(C)/C(=O)O)[C@H]1CC[C@@]2([C@@]1(CCC3=C2C(=O)C[C@@H]4[C@@]3(CCC(=O)C4(C)C)C)C)C</chem>		
Ganoderic acid Y	<chem>CC(CCC=C(C)C(=O)O)C1CCC2(C1(CC=C3C2=CCC4C3(CCC(C4(C)C)O)C)C)C</chem>	76537295	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/76537295/SDF?record_type=3d
Ganoderal A	<chem>CC(CCC=C(C)C=O)C1CCC2(C1(CC(=O)C3=C2CCC4C3(CCC(C4(C)C)O)C)C)C</chem>	72732279	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/72732279/SDF?record_type=3d
Ganoderol F	<chem>CC(CCC=C(CO)CO)C1CCC2(C1(CC=C3C2=CCC4C3(CCC(=O)C4(C)C)C)C)C</chem>	15602265	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/15602265/SDF?record_type=3d
Ganodermanondiol	<chem>C[C@H](CC[C@@H](C(C)(C)O)O)[C@H]1CC[C@@]2([C@@]1(CC=C3C2=CCC4[C@@]3(CCC(=O)C4(C)C)C)C)C</chem>	10253565	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/10253565/SDF?record_type=3d
Ganoleucinic acid E	<chem>CC(CCC=C(C)C(=O)O)C1CC(C2(C1(CC(=O)C3=C2CCC4C3(CCC(=O)C4(C)C)C)C)O</chem>	73198882	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/73198882/SDF?record_type=3d
Ganosporelactone B	<chem>CC1CC2(CC(C(=O)O2)C)C3C1C4(C(C(=O)C5=C(C4(C3=O)C)C(C6C5(CCC(C6(C)C)O)C)O)O)C</chem>	78384958	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/78384958/SDF?record_type=3d
Lucidone A (Luci)	<chem>CC(CCC=C(C)C=O)C1CCC2(C1(CC=C3C2=CCC4C3(CCC(C4(C)C)O)C)C)C</chem>	75069543	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/75069543/SDF?record_type=3d
Lucidone B (Luci)	<chem>CC(CCC=C(C)C=O)C1CCC2(C1(CCC3=C2C(=O)CC4C3(CCC(=O)C4(C)C)C)C)C</chem>	85116100	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/85116100/SDF?record_type=3d
Lucidone C (Luci)	<chem>CC(CCC=C(C)C=O)C1CCC2(C1(CCC3=C2C(=O)CC4C3(CCC(C4(C)C)O)C)C)C</chem>	85122815	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/85122815/SDF?record_type=3d
Ganoderone A	<chem>CC(=O)C1CC(=O)C2(C1(CC(=O)C3=C2C(C4C3(CCC(C4(C)C)O)C)O)C)C</chem>	14109413	https://pubchem.ncbi.nlm.nih.gov/rest/pug/compound/cid/14109413/SDF?record_type=3d

Target prediction of GLP

Through literature review, 43 experimentally verified targets of GLP were obtained, covering pathways such as PI3K/Akt/mTOR, TGF- β 1/Smad, TLR4/NF- κ B, AGE-RAGE, Nr2/HO-1, apoptosis regulation, and the RAS system. Among these, 11 targets overlapped with Track A, while 32 were unique to the GLP track. Merging the two tracks yielded a total of 200 drug targets.

Identification of common targets

After merging and deduplicating the results from the retrieval using the five keywords in the GeneCards database, a total of 10 918 disease-related targets were obtained. Intersection of the drug targets (200) with the disease targets yielded 184 common targets (Fig. 1), among which 143 targets were contributed by Track A, 30 targets by Track B, and 11 targets shared by both tracks.

PPI network construction and Hub gene screening

A PPI network was constructed using the 184 common targets (Fig. 2), comprising 182 nodes and 2 317 edges (including 2 288 high-confidence connections, 3 weak connections of hnRNPF, and 26 manually supplemented connections of VDR). Using degree ≥ 36 (twice the median degree value of 18) as the threshold, 47 hub genes were identified. The top 10 hub genes by the degree value were: IL6 (100), TNF (98), AKT1 (98), IL1B (93), PPARG (84), PTGS2 (84), CASP3 (77), ESR1 (76), BCL2 (76), and NFKB1 (71).

hnRNPF exhibited only weak connections in the PPI network, that is, the connections to TNF (score = 0.186), AKT1 (0.154), and HIF1A (0.176), all below the 0.4 confidence threshold.

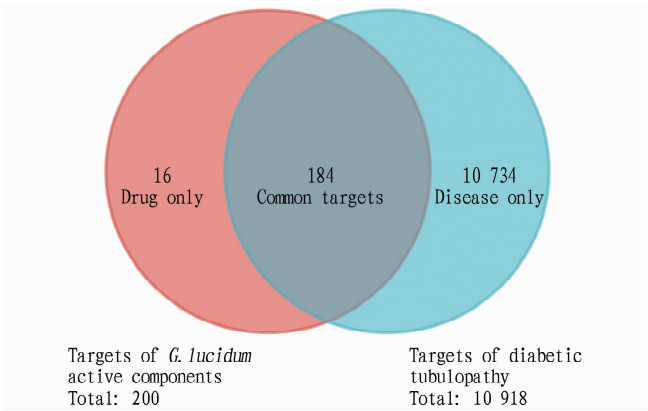
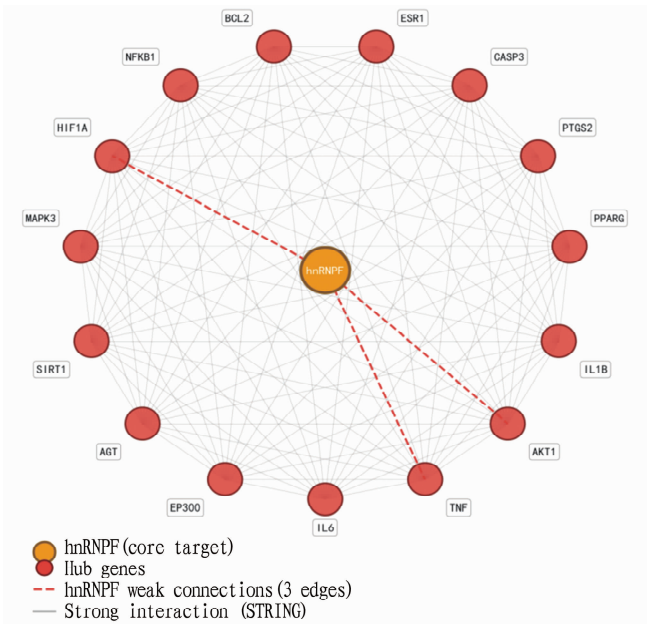


Fig. 1 Venn diagram of common targets between *G. lucidum* active components and diabetic tubulopathy



Although hnRNPF (golden) has only 3 weak connections (red dashed lines), it plays a core role in post-transcriptional regulation as an RNA-binding protein, and serves as a key node in the prevention and treatment of diabetic tubulopathy with *G. lucidum*.

Fig. 2 PPI network of common targets (182 nodes, 2 317 edges; red = hub genes, red dashed = hnRNPF weak connections)

GO functional annotation and KEGG pathway enrichment analysis

GO analysis identified 1 681 biological process (BP) terms and 214 molecular function (MF) terms (FDR <0.05). The top-ranked BP terms included Cellular response to oxygen-containing compound (GO:1901700, 108 genes), Cellular response to organic substance (GO:0010033, 120 genes), and Cellular response to endogenous stimulus (GO:0009719, 90 genes) (Fig. 3).

KEGG pathway enrichment analysis identified 178 significantly enriched pathways. Among the pathways associated with diabetic complications, the AGE-RAGE signaling pathway (hsa04933, 24 genes, FDR = 2.18×10^{-23}) was the most significantly enriched, followed by insulin resistance (hsa04931, 19

genes), the TNF signaling pathway (hsa04668, 16 genes), the HIF-1 signaling pathway (hsa04066, 15 genes), the PI3K-Akt signaling pathway (hsa04151, 28 genes), etc. (Fig. 4).

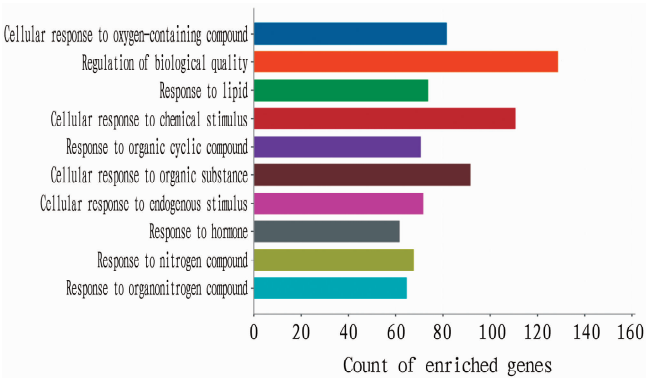


Fig. 3 GO biological process enrichment analysis (Top 10, deduplicated)

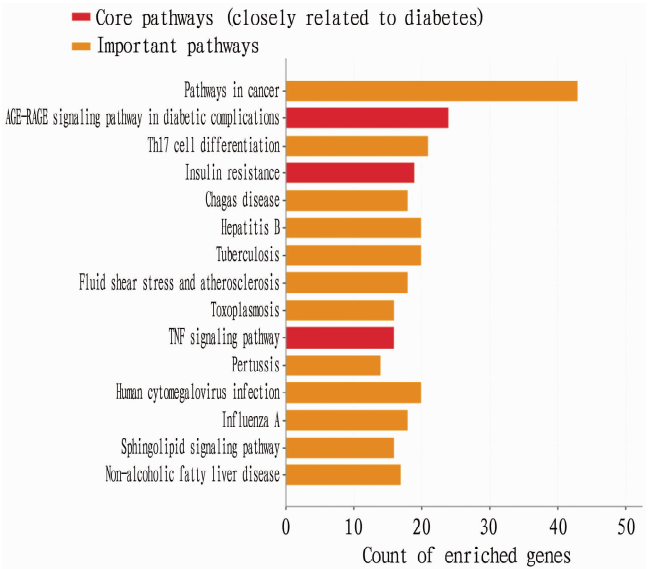


Fig. 4 KEGG pathway enrichment analysis (Top 20; red label = AGE-RAGE signaling pathway)

Molecular docking verification

Ten representative compounds were selected for docking with six target proteins (Table 2, Fig. 5). The binding energies of the 60 docking runs ranged from -0.55 to -13.27 kcal/mol.

A total of 35 docking runs exhibited binding energies ≤ -7.0 kcal/mol (strong binding activity). Ganosporelactone B showed the strongest binding to four targets; hnRNPF (-10.22 kcal/mol), HIF1A (-13.27), MTOR (-10.53), and TNF (-9.47). PPARG exhibited ligand type selectivity: triterpenoid aldehydes showed stronger binding (ganoderal B, -10.74; ganoderal A, -10.63), while triterpenoid acids showed weaker binding (ganoderic acid DM, -1.04).

All key components exhibited binding energies of < -4.25 kcal/mol with the key targets, indicating that all showed binding activity.

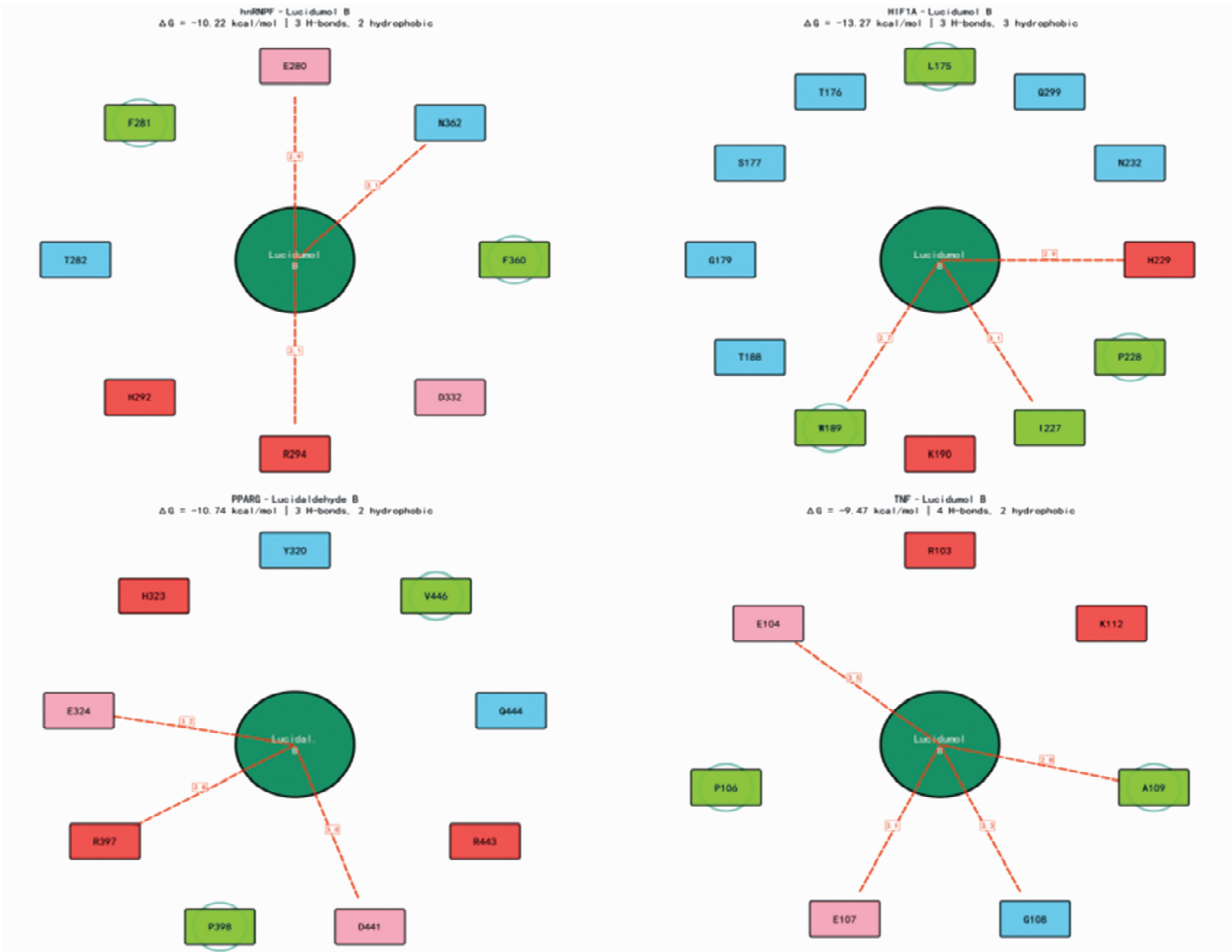


Fig. 5 2D interaction diagrams of molecular docking (4 representative complexes; red dashed lines = hydrogen bonds, green arcs = hydrophobic contacts)

Table 4 Molecular docking binding energy matrix (kcal/mol)

Compound	Type	AKT1	HIF1A	hnRNP	MTOR	PPARG	TNF
Ganosporelactone B	Lactone	-3.80	-13.27	-10.22	-10.53	-2.14	-9.47
Ganoderone A	Triterpenoid ketone	-6.71	-11.42	-9.62	-8.72	-2.06	-9.41
Ganoleucinic acid E	Triterpenoid acid	-5.86	-11.19	-9.25	-8.58	-1.62	-7.20
Lucidone B_Luci	Triterpenoid aldehyde	-6.16	-10.81	-8.49	-8.16	-10.74	-7.35
Ganoderol A	Triterpenoid aldehyde	-6.81	-10.43	-8.43	-8.20	-1.80	-7.45
Epoxyganoderiol B	Triterpenoid	-6.45	-10.97	-8.24	-8.17	-10.42	-7.02
Ganoderic acid Y	Triterpenoid acid	-6.31	-10.77	-8.06	-8.32	-1.59	-7.12
Ganoderol F	Triterpenoid alcohol	-5.80	-9.86	-7.28	-7.83	-1.21	-6.51
Ergostadienol	Steroid	-7.11	-9.89	-7.89	-7.77	-10.11	-6.81
β-Sitosterol	Steroid	-6.57	-7.38	-5.30	-5.51	-0.55	-3.93
Protein average							
Average binding energy		-6.16	-10.60	-8.28	-8.18	-4.22	-7.23

Bold values indicate strong binding affinity (≤ -7.0 kcal/mol).

Conclusion and Discussion

Through a dual-track strategy integrating TCMSp-based small-molecule screening and literature-based GLP target mining, a total of 200 drug targets were obtained, of which 32 were unique

to the GLP track. It indicates that literature mining provides complementary coverage of targets for polysaccharide components. Intersection with disease targets yielded 184 common targets, and the PPI network identified 47 hub genes, with inflammatory

cytokines such as IL6, TNF, AKT1, and IL1B serving as core nodes. In the KEGG enrichment analysis, the AGE-RAGE signaling pathway ranked first with 24 genes enriched and the lowest FDR. The high enrichment of this pathway has clear pathological significance. Specifically, chronic hyperglycemia promotes non-enzymatic glycation reactions of proteins, generating large amounts of advanced glycation end products (AGEs). Binding of AGEs to their receptor RAGE triggers the nuclear translocation and activation of the downstream transcription factor NF- κ B, which in turn drives the transcriptional upregulation of pro-inflammatory cytokines such as TNF- α and IL-6, as well as the accumulation of reactive oxygen species (ROS), ultimately leading to apoptosis of renal tubular epithelial cells and acceleration of interstitial fibrosis^[18–19]. The molecular docking results showed that Ganosporelactone B had a binding energy of -9.47 kcal/mol with TNF, and AGER and NOX4, which are also components of the AGE-RAGE pathway, were identified among the GLP literature targets, suggesting that the active components of *G. lucidum* may block this pathway at multiple nodes. It is noteworthy that previous studies on *G. lucidum* have mostly been limited to the single-pathway analysis of small-molecule triterpenoids, while polysaccharide macromolecules have long been avoided due to their structural complexity and difficulty in screening through conventional databases, resulting in a fragmented understanding of the overall pharmacologically active material basis of *G. lucidum*. In this study, the dual-track strategy was adopted to incorporate GLP literature targets into the network pharmacology analysis framework for the first time, filling this systematic gap and providing relatively comprehensive computational evidence for the multi-component, multi-target, and multi-pathway synergistic mechanisms of *G. lucidum* against diabetic tubulopathy.

In the PPI network, hnRNPF exhibited only weak connections with TNF, AKT1 (a core kinase of the PI3K/Akt pathway^[20]), and HIF1A (scores 0.154–0.186), which aligns with its biological characteristic as an RNA-binding protein—hnRNPF primarily regulates alternative splicing by recognizing G-tract sequences^[6], rather than relying on direct protein-protein interactions. Previous studies have shown that hnRNPF regulates the expression of apoptosis-related factors in renal tubular cells under diabetic conditions^[7] and plays a critical role in maintaining podocyte function^[8]. The molecular docking results showed that Ganosporelactone B exhibited a binding energy of -10.22 kcal/mol with hnRNPF, followed by ganoderone A (-9.62) and ganoleucic acid E (-9.25), suggesting that *G. lucidum* triterpenoids may directly bind to hnRNPF and interfere with its RNA-binding activity. The ligand selectivity of PPARC (strong binding of triterpenoid aldehydes, weak binding of triterpenoid acids) suggests that the aldehyde functional group may serve as a key structural basis for PPARC ligand recognition.

The present study has several limitations. (1) All target predictions were based on public databases, and algorithmic differences among databases may introduce systematic bias. (2) Molecular

docking was performed using semi-flexible docking, which does not account for protein conformational dynamics^[21]. (3) The AlphaFold-predicted structure of hnRNPF has not been experimentally validated. (4) The PPI network only reflects protein-level interactions and does not capture the post-transcriptional regulatory effects of hnRNPF. (5) The conclusions are based on computational predictions, which lack experimental verification. And (6) some triterpenoid compounds may contain PAINS (pan-assay interference compounds) structural alerts, requiring experimental verification to exclude false positives^[22]. Future studies could verify the actual binding affinity between candidate compounds and hnRNPF through surface plasmon resonance (SPR) or isothermal titration calorimetry (ITC) experiments, identify the RNA binding spectrum of hnRNPF via RIP-seq or CLIP-seq, and provide experimental support through *in vivo* pharmacodynamic studies.

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system for Emerging Engineering talent cultivation in food science is a key measure for ensuring talent cultivation quality and promoting continuous optimization of education and teaching. The system is built with multiple evaluation subjects including students, teachers, enterprises, alumni, and society, actively introduces third-party professional accreditation and assessment, and establishes a graduate career development database to track long-term cultivation outcomes. In terms of evaluation methods, the system implements a combination of formative assessment and summative assessment, emphasizes process-oriented assessment, introduces real enterprise projects to evaluate students' ability to solve complex engineering problems, and establishes student learning portfolios to record growth trajectories. The ultimate focus of the evaluation is continuous improvement, with its results serving as important bases for revising training programs, optimizing teaching, and allocating resources. A feedback mechanism is established to timely communicate results to all parties, and the effectiveness of Emerging Engineering Education is incorporated into the performance assessment of departments and faculty^[10].

Conclusions and Prospect

The development of Emerging Engineering Education provides a historical opportunity for the reform of food education, but also brings severe challenges. The talent cultivation model proposed in this article, which includes "five-dimensional synergy, four-chain integration, and tripartite convergence", aims to build a diversified collaborative education mechanism, connect the innovation chain of the education industry, and deepen the integration of industry, education, science and technology innovation. The development of Emerging Engineering Education is an ongoing journey. Guided by Xi Jinping Thought on Socialism with Chinese Characteristics for a New Era, universities adhere to the fundamental task of fostering virtue through education and continuously deepen educational and teaching reforms. This talent cultivation model aims to cultivate more outstanding engineers for the

highquality development of the food industry, and to thereby make greater contributions to the construction of a strong country in education, science and technology, and talent.

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