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Analysis of 'Component-Target-Pathway' and Mining of Potential Biomarkers for Yinchenhao Decoction Combined with Lamivudine in the Treatment of Alcoholic Liver Disease and Its Derived Disorders

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Abstract: *This study aimed to investigate the potential common targets, core active ingredients, and key signaling pathways of Yinchenhao Decoction combined with Lamivudine in the treatment of alcoholic liver disease (ALD) using network pharmacology and molecular docking, providing a theoretical basis for this integrative therapy. Active ingredients of Yinchenhao Decoction and Lamivudine targets were screened from the TCMSP and ChEMBL databases, while ALD-related targets were obtained from GeneCards, TTD, and DrugBank. Common targets were identified via Venny analysis, and a protein-protein interaction (PPI) network was constructed using STRING and Cytoscape. GO and KEGG enrichment analyses were performed with Metascape, and molecular docking was conducted using CB-Dock2. A total of 44 active compounds, 331 drug targets, and 1022 ALD targets were surveyed, yielding 44 overlapping targets. GO analysis indicated involvement in hormone response and nuclear receptor activity, while KEGG enrichment highlighted the "Chemical carcinogenesis–receptor activation" pathway. Molecular docking showed that aloe-emodin had the strongest binding affinity with IL-6 (–9.7), followed by lamivudine (–6.4), quercetin (–6.2), and kaempferol (–6.1). In conclusion, Yinchenhao Decoction combined with Lamivudine may treat ALD through multiple components acting on multiple targets and pathways, primarily via IL-6-driven inflammation control and nuclear receptor modulation, and IL-6 appears to be a useful biomarker for monitoring ALD progression under this combined therapy.*

Keywords: *Alcoholic liver disease; Yinchenhao Decoction; Lamivudine; Network pharmacology; Molecular docking; IL-6; Chemical carcinogenesis-receptor activation*

1. Introduction

Alcoholic liver disease (ALD) is a pathological condition of the liver brought on by prolonged or heavy drinking, which may involve stages—ranging from fatty liver or hepatitis to full-blown cirrhosis. For 2016 as a whole, alcohol was responsible for 3 million global deaths or 5.3% of all deaths[1]. The liver is the principal site of alcohol metabolism (roughly 90%of it) (with 2%- 10%being handled by the lungs or the kidneys), consider alcohol as one of the principal factors that disturb normal hepatic physiology. With regard to alcohol metabolism in the liver, alcohol dehydrogenase (ADH) acts to convert ethanol to acetaldehyde—a very reactive and harmful byproduct. Nowadays main approach to ALD treatment is to stop drinking in order to slow the disease's progress. As stated in the ' Guidelines for the Prevention and Treatment of Alcoholic Liver Disease ' (2018 revised edition), corticosteroids seem to be useful for improving short-term survival rates. Metadoxine promotes ethanol metabolism and eases the symptoms of alcohol intoxication. Still, such treatments do not always yield good results and have definite shortcomings—corticosteroids, for example, help little with long - term survival and may cause major side effects. For this reason, new therapeutic approaches deserve serious consideration.

Yinchenhao Decoction possesses hepatoprotective and choleretic effects and consists of three herbal components: Artemisia capillaris, Gardenia jasminoides, and Rheum palmatum. Studies have shown that Yinchenhao Decoction exerts a favorable hepatoprotective effect[2], and is currently mainly used for digestive and hepatobiliary system disorders. Lamivudine, a nucleoside analog antiviral drug, is primarily used to treat hepatitis B virus (HBV) and human immunodeficiency virus (HIV) infections. However, the common targets for Yinchenhao Decoction and Lamivudine in the treatment of ALD remain unclear.

Compared with the traditional 'single-target, single-drug' research mode, network pharmacology

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adopts a systems biology perspective. By constructing a multidimensional 'drug-component-target-disease' network, it can systematically reveal the 'multi-component, multi-target, synergistic' mechanisms of TCM and its decoctions, compensating for the limitations of traditional experimental methods in systemic depth and mechanistic breadth. Currently, research on ALD is largely confined to individual TCM formulas or Western medicine, lacking studies on the combination of both. The present work applies network pharmacology to evaluate the therapeutic targets, the molecules involved, and the signaling mechanisms of Yinchenhao Decoction and Lamivudine for ALD, thus laying down theory for such treatment approaches. Also considered is molecular docking as a tool to check the principal pathways and core targets, offering possible guidance for the treatment of ALD.

2. Materials and Methods

2.1 Screening of Active Ingredients and Targets of Yinchenhao Decoction

The Traditional Chinese Medicine Systems Pharmacology Database and Analysis Platform (TCMSP) (www.tcmsp-e.com) was used to retrieve the chemical constituents of the components of Yinchenhao Decoction (Artemisia capillaris, Gardenia jasminoides, Rheum palmatum). Active ingredients with an oral bioavailability (OB) ≥ 30% and drug-likeness (DL) ≥ 0.18 were selected for the study. The UniProt database was utilized to query and convert these into standard Gene Symbol nomenclature.

2.2 Mining of Lamivudine-Related Target Genes

The structural formula of Lamivudine was retrieved from the ChEMBL database (www.ebi.ac.uk/chembl), and its structural file was submitted to the PharmMapper database to identify drug targets. After screening, all obtained protein targets were input into the UniProt protein database to analyze and standardize their corresponding UniProt IDs, thereby identifying the targets associated with the combination of Yinchenhao Decoction and Lamivudine.

2.3 Screening of ALD-Related Targets

Using 'alcoholic liver disease (ALD)' as the keyword, disease-related targets were obtained from three databases: GeneCards (www.genecards.org), TTD (ttd.idrblab.cn), and DRUGBANK (go.drugbank.com). Duplicate entries were removed to identify the key therapeutic targets for ALD.

2.4 Construction of the Protein-Protein Interaction (PPI) Network

Venn diagrams were generated for the targets of Yinchenhao Decoction, Lamivudine, and ALD. The overlapping targets were submitted to the STRING 11.0 database (cn.string-db.org), with the species set to 'Homo sapiens' and a 'medium confidence' threshold > 0.4 set as the minimum interaction threshold. Disconnected protein targets were removed to construct the PPI network model and identify potential cellular functional modules.

2.5 Construction of the "Yinchenhao Decoction-Lamivudine-ALD" Target Network

The NCA plug-in in Cytoscape 3.12.0 was utilized to perform topological analysis of the PPI network-related targets based on the "Degree value," thereby constructing the "Yinchenhao Decoction-Lamivudine-ALD" target network.

2.6 Bioinformatics Analysis

The intersection targets of Yinchenhao Decoction, Lamivudine, and ALD were imported into the Metascape database (metascape.org/gp/#/main/step1) to acquire bioinformatics information. The top 5-10 items, ranked by P-value, were selected for Gene Ontology (GO) enrichment analysis and Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analysis. Information regarding biological processes (BP), cellular components (CC), molecular function (MF), and signaling pathways was obtained through GO and KEGG enrichment analyses. Subsequent visualization yielded enrichment analysis bar charts, bubble charts, and a Sankey diagram of the drug-active ingredient-target-pathway network.

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2.7 Molecular Docking Validation

The 3D structures of the key active ingredients of Yinchenhao Decoction and Lamivudine (quercetin, kaempferol, aloe-emodin, and Lamivudine), selected based on ranked Degree values, and the protein crystal structure of the key factor in ALD (IL6) were obtained from the ChEMBL database (www.ebi.ac.uk/chembl) and PDB database (www.rcsb.org), respectively. Molecular docking analysis for these component-target pairs was performed using the CB-Dock2 platform (cadd.labshare.cn).

3. Results

3.1 Screening of Active Ingredients and Targets of Yinchenhao Decoction

A total of 44 chemical constituents were screened from the ingredients of Yinchenhao Decoction, specifically 16 from Rheum palmatum, 13 from Artemisia capillaris, and 15 from Gardenia jasminoides (see Table 1, Table 2, and Table 3).

Table 1 Information on active ingredients of Rheum palmatum

MOL ID	Molecule Name	OB (%)	DL
MOL002235	EUPATIN	50.8	0.41
MOL002251	Mutatochrome	48.64	0.61
MOL002259	Physciondiglucoside	41.65	0.63
MOL002260	Procyanidin B-5,3'-O-gallate	31.99	0.32
MOL002268	Rhein	47.07	0.28
MOL002276	Sennoside E_qt	50.69	0.61
MOL002280	Torachrysone-8-O-beta-D-(6'-oxayl)-glucoside	43.02	0.74
MOL002281	Toralactone	46.46	0.24
MOL002288	Emodin-1-O-beta-D-glucopyranoside	44.81	0.8
MOL002293	Sennoside D_qt	61.06	0.61
MOL002297	Daucosterol_qt	35.89	0.7
MOL002303	Palmidin A	32.45	0.65
MOL000358	Beta-sitosterol	36.91	0.75
MOL000471	Aloe-emodin	83.38	0.24
MOL000554	Gallic acid-3-O-(6'-O-galloyl)-glucoside	30.25	0.67
MOL000096	(-)-Catechin	49.68	0.24

Table 2 Information on active ingredients of Artemisia capillaris

MOL ID	Molecule name	OB (%)	DL
MOL000354	Isorhamnetin	49.6	0.31
MOL000358	Beta-sitosterol	36.91	0.75
MOL004609	Areapillin	48.96	0.41
MOL005573	Genkwanin	37.13	0.24
MOL007274	Scrophulein	30.35	0.3
MOL008039	Isoarcapillin	57.4	0.41
MOL008040	Eupalitin	46.11	0.33
MOL008041	Eupatolitin	42.55	0.37
MOL008043	Capillarisin	57.56	0.31
MOL008045	4'-Methylcapillarisin	72.18	0.35
MOL008046	Demethoxycapillarisin	52.33	0.25
MOL008047	Artepillin A	68.32	0.24
MOL000098	Quercetin	46.43	0.28

Table 3 Information on active ingredients of Gardenia jasminoides

MOL ID	Molecule name	OB (%)	DL
MOL001406	Crocetin	35.3	0.26
MOL001663	(4aS,6aR,6aS,6bR,8aR,10R,12aR,14bS)-10-hydroxy-2,2,6a,6b,9,9,12a-heptamethyl-1,3,4,5,6,6a,7,8,8a,10,11,12,13,14b-tetradecahydropicene-4a-carboxylic acid	32.03	0.76

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MOL001941	Ammidin	34.55	0.22
MOL004561	Sudan III	84.07	0.59
MOL000098	Quercetin	46.43	0.28
MOL000358	Beta-sitosterol	36.91	0.75
MOL000422	Kaempferol	41.88	0.24
MOL000449	Stigmasterol	43.83	0.76
MOL001494	Mandenol	42.00	0.19
MOL001506	Supraene	33.55	0.42
MOL001942	Isoimperatorin	45.46	0.23
MOL002883	Ethyl oleate (NF)	32.4	0.19
MOL003095	5-hydroxy-7-methoxy-2-(3,4,5-trimethoxyphenyl)chromone	51.96	0.41
MOL007245	3-Methylkaempferol	60.16	0.26
MOL009038	GBGB	45.58	0.83

3.2 Lamivudine and ALD Related Target Screening

Through database retrieval and screening, a total of 341 potential lamivudine targets were initially obtained, and 280 unique drug targets were acquired after removing duplicates. Simultaneously, 1022 key ALD-related targets were identified by integrating data from GeneCards, TTD, and DRUGBANK databases.

3.3 PPI Network Analysis of Key Targets for Yinchenhao Decoction and Lamivudine in the Treatment of ALD

The screened targets of Yinchenhao Decoction and Lamivudine were intersected with alcoholic liver disease targets. As shown in Figure 1, there are 51 overlapping targets between Yinchenhao Decoction and Lamivudine, 108 overlapping targets between Yinchenhao Decoction and alcoholic liver disease, and 154 overlapping targets between Lamivudine and alcoholic liver disease. Nodes represent target proteins, and each edge represents the interaction between target proteins; the degree value of each target reflects its significance within the entire network. From Figure 2 it may be seen that—in the Cytoscape network built here—targets are arranged by descending degree value (from outer to inner concentric circles) with an increasing link to alcoholic liver disease. According to this degree-based analysis, the principal target proteins in the network are ALB, IL6, and TNF. Such targets and their molecular roles appear to be central to the combined therapeutic effect of Yinchenhao Decoction and Lamivudine upon alcoholic liver disease.

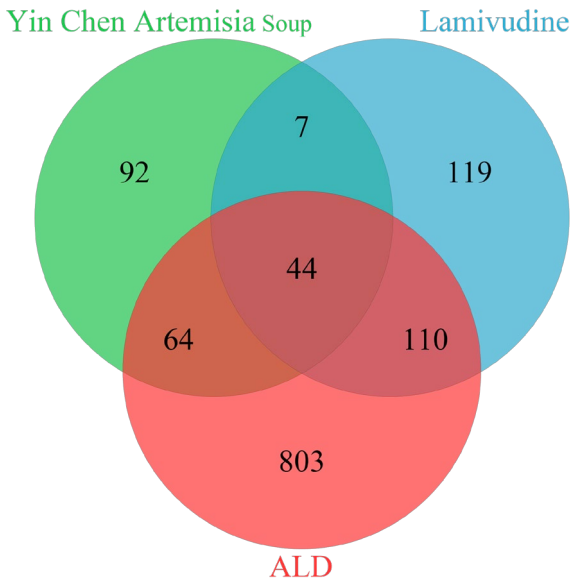

Figure 1 Intersection of targets among Yinchenhao Decoction, Lamivudine, and ALD

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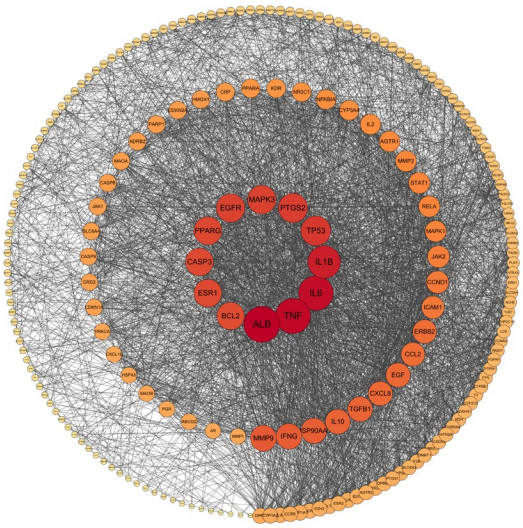

Figure 2 PPI network of key target interactions

3.4 Results of bioinformatics analysis (GO and KEGG pathway enrichment analysis)

GO enrichment analysis has been applied to determine the biological functions, molecular roles, and parts of the cell that are involved in the combined treatment of alcoholic liver disease (ALD) by means of Yinchenhao Decoction and Lamivudine. According to Figure 3, the combination seems to act mainly upon such biological processes as response to hormone or to organic cyclic compound or to lipid-containing substances. Its functions are carried out in cellular locations—specifically the membrane raft, membrane microdomain, or caveola. Among the microscopic phenomena it controls are nuclear receptor actions, ligand - dependent transcription factors, and nuclear steroid receptors. With regard to KEGG pathway analysis of the joint treatment of ALD with Yinchenhao Decoction and Lamivudine, the relevant pathways were defined by means of P-value analysis. From the standpoint of Figure 4,the combination therapy is of considerable importance for the Chemical carcinogenesis-receptor activation pathway in the treatment of ALD.

With regard to the "component-target-mechanism-pathway" network pharmacology analysis, the interaction of integrated Chinese and Western medicines with their targets and with various pathways has been clarified. As shown in Figure 5 and Table 1, the combined treatment of Yinchenhao Decoction with Lamivudine brings about therapeutic results by virtue of action upon targets including ALB, IL-6, and TNF, and upon substances like quercetin, arachidonic acid, formononetin, beta - sitosterol, or prednisolone, as well as upon pathways—such as Chemical carcinogenesis-receptor activation—which may influence microbial or macroscopic phenomena.

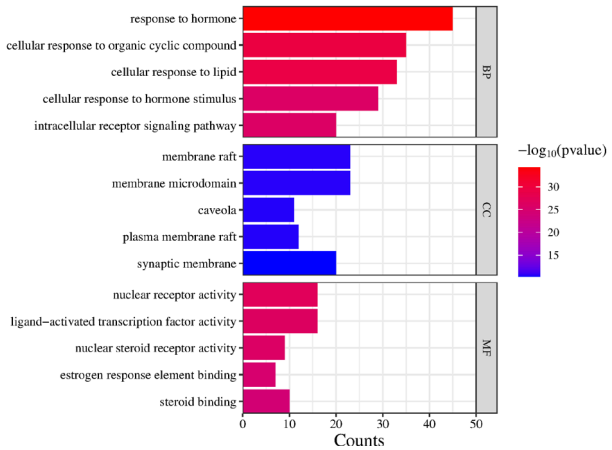

Figure 3 GO analysis of the combined treatment of ALD with Yinchenhao Decoction and Lamivudine

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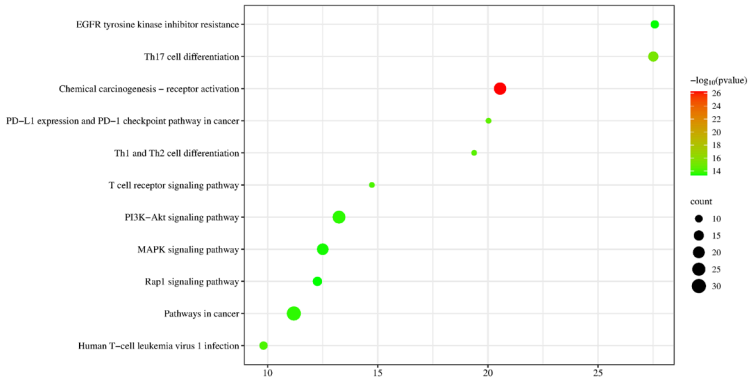

Figure 4 GO analysis of the combined treatment of ALD with Yinchenhao Decoction and Lamivudine

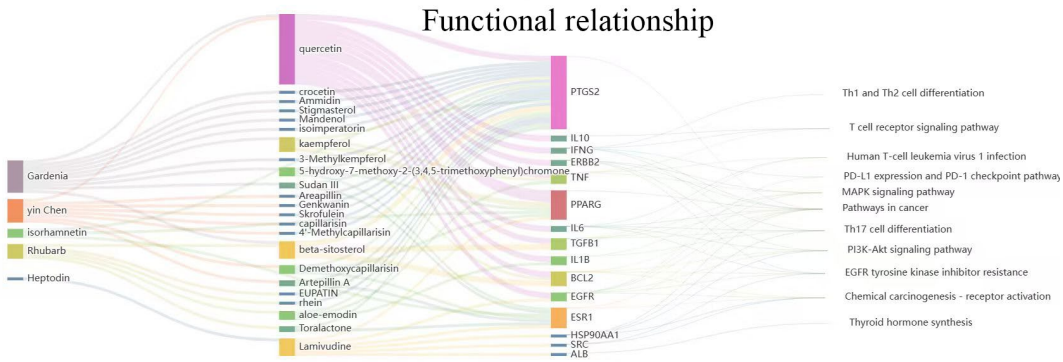

Figure 5 Sankey-bubble diagram of targets and pathways for the combined treatment of ALD with Yinchenhao Decoction and Lamivudine

3.5 Molecular docking validation of key active ingredients of drugs and key targets of ALD

Molecular docking was performed between the potential major active ingredients of the Yinchenhao Decoction-Lamivudine combination (quercetin, kaempferol, aloe-emodin, and lamivudine) and the core target IL6. As shown in Table 4, the major active ingredients exhibited varying affinities for the core target, with the highest affinity observed for aloe-emodin-IL-6, showing a binding energy of -9.7 kJ/mol. The binding conformation of the active ingredients with the target is presented in Figure 6.

Table 4 Binding affinity of active ingredients to targets

Active ingredients	Core target	Binding affinity (eV)
Quercetin	IL-6	-6.2
Kaempferol		-6.1
Aloe-emodin		-9.7
Lamivudine		-6.4

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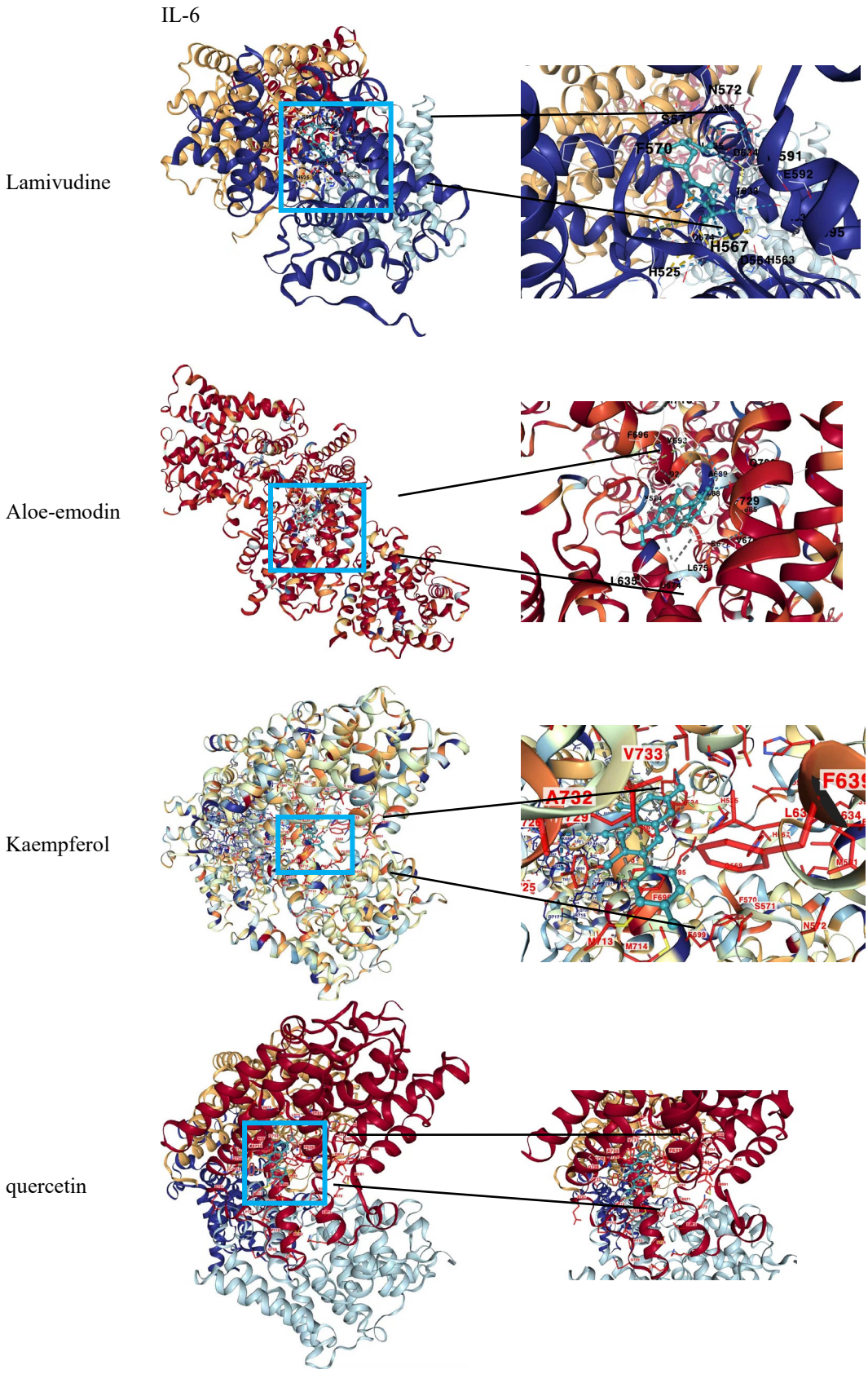

Figure 6 Molecular docking results of IL-6 with key active ingredients

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4. Discussion

With rapid economic development and improved living standards, alcohol consumption has increased annually. For the past ten years or so, the per capita consumption of pure alcohol—among persons aged 15 and above—in China has gone up by 76% to 6.0 L, which is above the world average. As a result, the proportion of people with chronic liver disease (mainly ALD) has grown very fast: from 27.0% to 66.2% in fifteen years[3]. Alcohol intake has a positive correlation with liver injury and may be categorized—according to the degree of lesion—as alcoholic fatty liver, alcoholic hepatitis, alcoholic fibrosis of the liver, alcoholic cirrhosis, or hepatocellular carcinoma. Cirrhosis kills about 610,000 people every year. Of all cirrhosis cases in developed countries, 60% are linked to alcohol, whereas in the Asia-Pacific area alcohol accounts for 25% of chronic liver disorders—a major threat to population health and to safety[1].

At present, ALB is part of the therapeutic approach to a number of liver disorders (such as cirrhosis, hepatic encephalopathy, or hepatorenal syndrome)[4]. Albumin plays an important role in the major prognostic scoring systems now applied to cirrhosis and is one of the basic prognostic indicators for such patients[5]. There is strong evidence that albumin infusion markedly raises the probability of short-term survival among those with decompensated cirrhosis and acute liver failure[6]. This study also treats ALB as one of the major possible targets for therapeutic use against alcoholic liver disease; targeting ALB is likely to be of central importance in controlling the tendency of alcohol - related inflammation to develop into fibrosis or cirrhosis. TNF is regarded as a basic pro-inflammatory cytokine in the context of liver pathology—one that may lead to injury or to hepatic failure. But, according to Kumar R et al., pentoxifylline and etanercept yield no substantial improvement in survival (and even raise infection rates); hence, they are excluded from further discussion[7].

Available research shows that the circulating IL-6 levels go up gradually as cirrhosis develops[8], and this seems to indicate a positive correlation between IL6 abundance and the severity of liver disease, Of particular note is the finding by Liu et al. that the IL-6 cytokine group is centrally involved in the pathogenesis of metabolic-associated fatty liver disease (MAFLD). Mechanistically, these cytokines seem to promote disease progression through disruption of lipid homeostasis or stimulation of lipid buildup, which in turn leads to hepatic steatosis and fastened fibrosis. Targeted suppression of the IL-6 signaling pathway is helpful in preventing liver damage and in slowing the course of illness. With regard to particular mechanisms, blocking the downstream JAK/STAT3 cascade appears to be highly effective against hepatocellular carcinoma—its proliferation and growth, this signaling axis is being highlighted as a possible therapeutic target[9]. From an overall standpoint, the findings establish IL6 as one of the major factors involved in liver disease progression. The present work identifies IL6 as a central target—among several—when treating ALD with Yinchenhao Decoction and Lamivudine, offering fresh ideas about how to assess the value of IL6 for ALD or for other unknown mechanisms of action.

With regard to enrichment analysis, the main pathway called Chemical carcinogenesis-receptor activation has been found by P-value filtering. Analysis of the central targets shows that they may be involved—among other things—in responses to hormones or to organic cyclic compounds, or in nuclear receptor function and in ligand-driven transcription.

From the standpoint of molecular docking it is evident that quercetin, kaempferol, aloe – emodin, and lamivudine attach themselves to the targets by means of hydrogen bonds, with very good binding energies and strong affinities, making them likely candidates for use in ALD; hydrogen bonding seems to be the principal form of interaction between the protein targets and the small-molecule agents. Available evidence suggests that quercetin plays it plays an important part in anti-inflammatory, antioxidant, antibacterial, and anticancer phenomena and may help to prevent or relieve alcohol-induced liver damage through lysosome-related actions[10, 11]. Kaempferol is known to show useful anticancer properties—it suppresses lipid formation in AFLD cells[12]—and is hepatoprotective because it limits proinflammatory cytokine production[13]. Aloe-emodin can, to some extent, amelioration of non-alcoholic fatty liver disease may be achieved by restoring ZNRF3 expression and by suppressing Wnt/β-catenin - mediated adipogenesis in the liver[14], but there are no reports detailing its effects on alcoholic liver disease. Lamivudine has proven to be useful for treating patients with hepatitis B-linked liver failure—by lowering serum HBV DNA values, bringing about normalization of ALT, and reducing hepatic inflammation to a marked degree. As an alternative treatment, in combination with silymarin, it can prevent or slow fibrosis in alcohol-induced HBV transgenic mice through regulation of the TGF - β1/Smads pathway, with lower CTGF expression and less stellate cell activation—thus helping to protect the organ[15].

Conventional in vitro cellular assays and in vivo animal studies are basic elements of pharmacological analysis but suffer from a variety of drawbacks—high cost, long time requirements, ethical problems

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with animal treatment—and are often responsible for many late-stage failures of candidate drugs(due to safety or efficacy issues). Yet computational simulations too come with their own restrictions, such as the usual neglect of total receptor flexibility, oversimplification of solvation effects, faults in empirical scoring functions, difficulty in determining exactly which compounds are agonists or antagonists, and the risk of false-positive hits—i.e., compounds with very good docking scores that do not work well later in trials or in practice. Thus molecular docking is best viewed as an initial, exploratory tool—not necessarily the conclusive determinant of biological behavior—and its positive results need to be tested independently by means of lab and animal experiments.

5. Conclusion

This study utilized network pharmacology and molecular docking methods to conduct an in-depth investigation into the key targets and mechanisms of the Yinchenhao Decoction-Lamivudine combination in the treatment of ALD from the perspective of "drug-active component-target-pathway." The study identified key targets including ALB, IL6, and TNF, as well as the significant pathway of Chemical carcinogenesis - receptor activation, and established IL6 as a biomarker for monitoring ALD, providing a clear direction for further experimental validation.

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