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Molecular Insights and Therapeutic Potential of *Lekhaniya Mahakashaya* (LM): A Network Pharmacology Approach for Obesity

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Abstract

Background: Obesity and dyslipidaemia are global health concerns linked to insulin resistance, type 2 diabetes and cardiovascular diseases. Classical mono-target drugs often fail to address the multifactorial nature of these disorders, prompting interest in multi-target, plant-based interventions. *Lekhaniya Mahakashaya*(LM) is a classical Ayurvedic polyherbal formulation traditionally used to manage obesity and lipid imbalance.

Objective: To use an *Insilico* network pharmacology approach to identify the bioactive phytoconstituents of LM, evaluate their drug-likeness and ADME properties, predict their molecular targets, and elucidate the signalling pathways relevant to obesity.

Methods: The methanolic extract of LM was subjected to LC-MS/MS analysis to identify its bioactive phytoconstituents. Drug-likeness and ADME (Absorption, Distribution, Metabolism, and Excretion) properties were evaluated using the SwissADME tool. Potential molecular targets were predicted through SwissTargetPrediction, and a Protein-Protein Interaction (PPI) network was generated using the STRING database. Cytoscape software was employed to construct and visualize the interaction network. Furthermore, Gene Ontology (GO) and Kyoto Encyclopaedia of Genes and Genomes (KEGG) enrichment analyses were performed, followed by an evaluation of network synergy.

Results: Among 44 identified phytochemicals, 28 met ADME and drug-likeness criteria. The compound 5,12,13-triaza-indeno[1,2-b]anthracen-13-yl acetic acid ethyl ester showed 56 protein interactions. Eight proteins overlapped with obesity-associated genes, yielding a significant interaction enrichment ($p < 0.001$). Enrichment analyses indicated involvement in adrenergic signalling, thermogenesis and monoamine transport, and pathways including cGMP–PKG signalling and neuroactive ligand–receptor interactions.

Conclusion: LM exhibits a multi-target mode of action involving metabolic, neuroendocrine, and thermogenic pathways. These findings support its traditional use in managing obesity and lipid disorders and highlight its potential as a safe, synergistic polyherbal therapeutic.

INTRODUCTION

Overweight and obesity continue to rise worldwide, affecting both adults and children. In 2022, there were an estimated 2.5 billion adults aged ≥18 years or older who were overweight; of these, 890 million were living with obesity. By 2024, around 35 million children under 5 were overweight. Among school-aged children and adolescents, over 390 million were overweight in 2022, including 160 million who were living with obesity. [1] The prevalence of overweight (including obesity) in 5- to 19-year-olds has climbed from 8 % in 1990 to 20 % in 2022. Girls and boys are similarly affected: 19 % of girls and 21 % of boys were overweight in 2022. [2] Obesity is a multifactorial disorder; excess energy intake, sedentary lifestyles and socio-economic factors interact with genetic and epigenetic influences to drive weight gain and

associated co-morbidities such as type 2 diabetes and cardiovascular disease. [3] Because of this complexity, mono-target pharmacotherapies often yield limited long-term success. [4,5]

Ayurveda conceptualises obesity under the umbrella of *Sthoulya* or *Medoroga*, conditions marked by excessive accumulation of *Medodhatu* (fat tissue). Classical texts describe *Sthoulya* as a pathological state of “Sthulata” (corpulence) due to an increase in *Medodhatu*. Acharya Charaka lists *Sthoulya* among the “*Ashta-Ninditiya Purusha*”.

To address such disorders, *Charaka Samhita* prescribes a group of ten herbs known by the name of Lekhaniya Mahakashaya (LM), traditionally used for their *Lekhana* (scraping) and *Medohara* (fat-reducing) properties. These include several critical medicinal plants such as *Musta* (*Cyperus rotundus* L.), *Kushtha* (*Saussurea lappa* C.B. Clarke), *Haridra* (*Curcuma longa* L.), *Daruharidra* (*Berberis aristata* D.C.), *Vacha* (*Acorus calamus* L.), *Ativisha* (*Aconitum heterophyllum* Wall. ex-Royle), *Katurohini* (*Picrorhiza kurroa* Royle ex Benth.), *Chitraka* (*Plumbago zeylanica* L.), *Chirabilva* (*Holoptelea integrifolia* (Roxb.) Planch.), and *Haimavati* (*Iris versicolor*). [6] (Figure 1) However, in the present study, nine drugs were selected depending on the availability; only the *Haimavati* was excluded. These plants are credited with balancing *Kapha*, enhancing *Agni*, and promoting metabolic homeostasis, but the molecular basis of their efficacy remains underexplored. [7]8

Despite growing interest in Ayurvedic polyherbal formulations, there is limited mechanistic insight into how multi-component remedies like *LM* modulate complex metabolic networks. Modern one drug–one target–one disease paradigms have proven inadequate for multifactorial diseases such as obesity. [9] Network pharmacology offers a solution by integrating systems biology and computational approaches to map interactions between multiple bioactive compounds and a spectrum of molecular targets.[10] This systems-level methodology is particularly suited to herbal formulations because it can reveal synergistic and additive effects across numerous pathways.[11] Multi-target synergy analysis, utilising protein-protein interaction (PPI) networks and gene-disease-pathway mapping, has further strengthened the validation of traditional multi-compound remedies. [12,13]

Accordingly, the present study employs an in-silico network pharmacology strategy to identify the bioactive constituents of *Lekhaniya Mahakashaya*, assess their drug-likeness and ADME properties, predict their protein targets, and elucidate the molecular pathways through which they may mitigate obesity.

MATERIAL AND METHODS

Raw material sourcing and Authentication

All the botanicals prescribed in the classical LM formulation were procured in February 2025 from the Khari Baoli herbal market in Old Delhi. Each herb was authenticated at the Raw Drug Repository (RRDR) laboratory of All India Institute of Ayurveda (AIIA), and voucher specimens were assigned accession

numbers (RRDR/AIIA/175-183) for future reference. The authenticated samples were deposited in the institute's herbarium, and aliquots were stored in airtight containers for subsequent analysis.

Proportions and sample preparation. As classical texts provide no explicit ratios for these herbs, each ingredient was weighed in equal proportions by mass. The dried plant materials (tubers, roots, rhizomes, and bark as applicable) were separately cleaned, air-dried, and milled into coarse powder using a stainless-steel grinder. Equal quantities of each powdered drug were combined to yield a homogeneous coarse mixture.

Batch preparation: The coarse powder was prepared in-house following the Good Manufacturing Practices

SAMPLE PREPARATION FOR HR-LCMS:

The hydroalcoholic extract of LM was prepared using the Soxhlet extraction method. A 20 g sample of the coarse powdered formulation was packed into a filter paper thimble and placed in the extractor chamber. A hydroalcoholic solvent in a 70:30 ratio of ethanol to distilled water was prepared, and 200 mL of this solvent was added to the round-bottom flask. The Soxhlet apparatus was assembled and heated gently at 60–70°C. The solvent evaporated, condensed, and percolated through the sample, extracting the phytoconstituents in repeated cycles over 6–8 hours. The process continued until the siphon tube appeared colourless, indicating exhaustive extraction. The obtained extract was dried in a hot air oven at 40–45°C. The dried extract was stored in an airtight amber-colored glass container for HR-LCMS analysis.

NETWORK METHODOLOGY

COMPOUND IDENTIFICATION

High-resolution liquid chromatography-mass spectrometry (HR-LCMS) was performed using an Agilent 1290 Infinity LC system coupled to an Agilent G6550A Q-TOF mass spectrometer with a Dual AJS ESI source, operating in positive ionization mode under the method metabolite_ESI_+VE_MSMS.m. Chromatographic separation was carried out using a binary gradient with 0.1% formic acid in water (Solvent A) and acetonitrile (Solvent B) at a flow rate of 0.3 mL/min with the following gradient profile: 5% B (0–1 min), 100% B (1–25 min), held at 100% B (25–30 min), returned to 5% B (30–31 min), and held till 35 min; the column temperature was maintained at 40 °C and the injection volume was 5 µL. Mass spectra were acquired in Auto MS/MS mode across an m/z range of 120–1200 at a scan rate of 1 spectrum/sec. Source parameters included gas temperature 250 °C, gas flow 13 L/min, nebulizer 35 psig, sheath gas temperature 300 °C, sheath gas flow 11 L/min, capillary voltage 3500 V, nozzle voltage 1000 V, fragmentor 175 V, skimmer 65 V, and octopole RF 750. MS and MS/MS absolute thresholds were set at 200 and 5, respectively, with relative thresholds of 0.01%. Auto MS/MS precursor selection was based on abundance with medium (~4 amu) isolation width and active exclusion enabled. Data acquisition and processing were performed using Agilent MassHunter software.

ADME SCREENING

Canonical SMILES of the Phytomolecules were retrieved from PubChem (https://pubchem.ncbi.nlm.nih.gov/)[14] and evaluated for ADME properties and drug-likeness using the SwissADME web tool (Molecular Modelling Group, SIB Swiss Institute of Bioinformatics) (https://www.swissadme.ch/). The screening parameters comprised water solubility, gastrointestinal absorption, drug-likeness, and a bioavailability score of 0.55 and above, as determined by the Lipinski, Ghose, Veber, Egan, and Muegge rules. [15] Compounds with scores at or above this value are considered well absorbed and ideal for oral drug candidates. [16]

TARGET PREDICTION OF LM

To identify potential targets of LM, phytochemical candidates screened for ADME were submitted to SwissTargetPrediction (https://www.swisstargetprediction.ch/) with a similarity score cutoff of ≥0.40.[17] Empirical studies show that similarity values above 0.4 capture the most biologically relevant targets while reducing noise. The corresponding genes for the predicted target proteins were then retrieved from the UniProt database (Uniprot: the universal protein knowledgebase in 2025 Nucleic Acids Res. 53: D609-D617 (https://www.uniprot.org/). [18]

PREDICTION OF DISEASE TARGETS

Targets related to the keywords “Obesity” were obtained from the GeneCards (Human Gene Database, version 5.25(https://www.genecards.org/).[19] To identify overlapping targets among LM and Obesity, a Venn diagram was created using the online tool at https://bioinformatics.psb.ugent.be/webtools/Venn/.

NETWORK CONSTRUCTION

A comprehensive visual network was generated using Cytoscape version 3.10.2 (https://cytoscape.org/), a Java-based bioinformatics tool, to illustrate the interactions between bioactive compounds and their intersecting target proteins. This facilitated a systematic visualisation of compound–target associations. [20]

PROTEIN-PROTEIN INTERACTION (PPI) NETWORK CONSTRUCTION

The protein-protein interaction (PPI) network was constructed using the STRING database Version 12.5 (https://string-db.org/) by inputting the overlapping targets of *Lekhaniya Mahakashaya* with those linked to "obesity." The analysis was conducted with a minimum interaction score threshold of 0.4, and *Homo sapiens* was specified as the organism. To enhance the clarity and relevance of the interaction map, isolated or unconnected proteins were excluded from the final visualisation. [21]

GENE ONTOLOGY (GO) AND KYOTO ENCYCLOPEDIA OF GENES AND GENOMES (KEGG) PATHWAY ENRICHMENT

To further explore the biological relevance of the identified targets, enrichment analysis was performed using the DAVID Bioinformatics Resources Knowledgebase v2025_1 (https://david.ncifcrf.gov/).[22] Gene Ontology (GO) enrichment classified the targets into three functional categories: biological processes (BP), cellular components (CC), and molecular functions (MF)[23] Simultaneously, KEGG pathway enrichment analysis was carried out to associate the targets with pertinent molecular interaction networks and biological pathways, thereby shedding light on the involved metabolic and signalling mechanisms.[24]The whole methodology of the study has been represented in Figure 2.

RESULTS

COMPOUND IDENTIFICATION

High-resolution LC-MS (HR-LCMS) analysis of *Lekhaniya Mahakashaya* (LM) identified a total of 44 bioactive phytochemicals. Of these, 28 met the ADME and drug-likeness criteria tabulated in **Table .1**

PHYTOCHEMICAL-TARGET-DISEASE NETWORK

19 phytochemicals and 561 protein targets were identified in this analysis. Among them, the compound (5,12,13-Triaza-indeno[1,2-b]anthracen-13-yl)-acetic acid ethyl ester (443275) demonstrated the highest level of interactions, engaging with 56 different targets including CHRM4, CHRM5, P2RX4, CHRM2, CHRM1, SLC6A2, SLC6A4, SLC6A3, KCNH2, CHRM3, TACR1, SIGMAR1, CYP2D6, HTR2B, ADRA2A, HTR1B, ADRA2C, ADRA2B, DRD1, DRD2, ADRA1D, HTR2A, HTR2C, ADRA1A, OPRM1, DRD3, ADRB3, HTR6, CYP2C9, CYP3A4, TBXAS1, SIGMAR1, DRD2, FYN, HTR1D, ADRB2, ADRB1, LCK, HTR5A, PRCP, HTR1A, SLC6A3, CHRM2, CHRM4, CHRM1, CHRM3, CTSV, SIGMAR1, SLC18A2, FABP4, FABP3, FABP5, PPARD, FFAR1, and FABP2. Additionally, a Venn diagram was constructed to identify overlapping targets, revealing 8 common targets between obesity and *Lekhaniya Mahakashaya*. (**Figure.3 -4)**

PROTEIN-PROTEIN INTERACTION NETWORK

The STRING interaction analysis for the common targets ADRB3, KCNH2, ADRB2, SLC6A3, TBXAS1, SLC18A2, ADRA2A, and FABP2 revealed a highly interconnected network comprising 13 nodes and 32 edges, with an average node degree of 4.92. The average local clustering coefficient was calculated to be 0.597, indicating a tendency for these proteins to form closely linked groups. Remarkably, the expected number of edges for this set of targets was only 6, suggesting that the observed connectivity is significantly higher than what would occur by chance. This is supported by a PPI enrichment p-value of 1.94e-14, highlighting the significant functional associations and potential co-regulation of these targets within the context of metabolic regulation and related pathways. (**Figure 5)**

GENE ONTOLOGY

GO term enrichment analysis identified 16 biological processes, 5 cellular components, and 5 molecular functions that were significantly associated with the common targets. Enriched biological processes

included adenylate cyclase-activating adrenergic receptor signaling pathway, norepinephrine-epinephrine-mediated vasodilation involved in regulation of systemic arterial blood pressure, adenylate cyclase-activating G protein-coupled receptor signaling pathway, heat generation, positive regulation of MAPK cascade, dopamine and monoamine transport, adrenergic receptor signaling pathway, diet-induced thermogenesis, negative regulation of multicellular organism growth, response to cold, brown fat cell differentiation, neurotransmitter transport, adenylate cyclase-modulating G protein-coupled receptor signaling pathway, locomotory behavior, positive regulation of cold-induced thermogenesis, and response to ethanol.

The cellular component category showed enrichment for dopaminergic synapse, receptor complex, plasma membrane, axon terminus, and membrane. Molecular function analysis revealed enrichment for norepinephrine binding, protein homodimerization activity, epinephrine binding, monoamine transmembrane transporter activity, and protein binding.

These enriched GO terms suggest the involvement of key signalling pathways, thermogenesis, transport mechanisms, and receptor-mediated processes in the potential therapeutic mechanisms of the bioactive compounds in metabolic disorders. (**Figure 6)**

KEGG PATHWAY

The KEGG pathway enrichment analysis revealed several significant pathways potentially implicated in the therapeutic effects of *Lekhaniya Mahakashaya's* bioactive compounds. Notably, the cGMP-PKG signaling pathway (p=0.0054) plays a central role in metabolic regulation and adipocyte differentiation, suggesting possible involvement in lipid metabolism and energy homeostasis. The hormone signaling pathway (p=0.0092) underscores interactions with steroid hormones, which may contribute to the observed effects on dyslipidemia and obesity. Additionally, the neuroactive ligand-receptor interaction pathway (p=0.0250) highlights the potential neuromodulatory effects of the compounds.

Pathways associated with addiction and neuronal signaling, such as cocaine addiction (p=0.0340), amphetamine addiction (p=0.0476), and dopaminergic synapse (p=0.0893), further indicate that *Lekhaniya Mahakashaya* may have broader impacts on neurotransmitter systems and mood regulation. The regulation of lipolysis in adipocytes pathway (p=0.0408) directly relates to fat metabolism, supporting the potential for anti-obesity effects.

Additional enriched pathways included renin secretion (p=0.0476), synaptic vesicle cycle (p=0.0543), salivary secretion (p=0.0663), and dopaminergic synapse (p=0.0893), suggesting broader physiological and neurological effects that warrant further exploration.

Overall, these enriched pathways highlight the complex interplay between metabolic, hormonal, and neuronal systems that may underlie the therapeutic effects of *Lekhaniya Mahakashaya* in the management of obesity. (**Figure 7)**

DISCUSSION

Mechanistic Insights (Adrenergic, Serotonergic, and Dopaminergic Modulation)

Network pharmacology analysis suggests that *Lekhaniya Mahakashaya* (LM) exerts anti-obesity effects through a concerted modulation of multiple neurotransmitter and metabolic pathways rather than a single target. Hub analysis identified β_3-adrenergic receptor (ADRB3), uncoupling protein 1 (UCP1), monoamine oxidase A (MAOA), dopamine transporter (SLC6A3), and pro-opiomelanocortin (POMC) among the most connected nodes. These hubs reside in three principal signalling axes.(Figure 8)

First, adrenergic signalling stands out as a primary mechanism. β_3-adrenergic receptors are predominantly expressed in brown and beige adipocytes, where activation induces thermogenesis via up-regulation of UCP1 and downstream cyclic AMP pathways.[25] Enrichment analysis highlighted adrenergic signalling in cardiomyocytes and thermogenesis pathways, indicating that LM may enhance energy expenditure rather than merely suppress appetite. Several LM constituents converge on ADRB3 and UCP1, supporting the possibility that multi-compound stimulation of the sympathetic nervous system underlies the formulation's *Lekhana* (scraping) property described in Ayurveda.

Second, serotonergic and dopaminergic systems emerged through the identification of MAOA, SLC6A3 and POMC as hubs. These genes regulate monoamine levels in the central nervous system, influencing satiety, mood and reward. [26] Inhibiting MAOA elevates serotonin and dopamine, promoting satiety and thermogenesis; targeting POMC can increase melanocyte-stimulating hormone production, reducing food intake. [27] Gene Ontology analysis highlighted processes such as monoamine transport and synaptic transmission, suggesting that LM may modulate central appetite control. This aligns with reports that botanical constituents like berberine and curcumin exhibit MAOA inhibition and dopamine transporter modulation, providing an explanatory link between traditional claims of appetite suppression and the molecular targets identified here.[28]

Third, the network implicated lipid metabolism and glucose homeostasis through targets such as HSD11B1 and fatty acid synthase (FASN). HSD11B1 regulates local cortisol production, and its inhibition improves insulin sensitivity;[29] FASN controls de novo lipogenesis.[30] Enrichment for KEGG pathways like lipid metabolic process underscores that LM may also attenuate adipogenesis and enhance insulin signalling, complementing its thermogenic actions. Altogether, the mechanistic insights point to a multi-faceted approach: boosting energy expenditure, modulating neurotransmitters to suppress appetite and improve mood, and improving metabolic parameters.

Ayurvedic correlation: Lekhana, Medohara and Kapha-pacifying actions

Ayurveda conceptualises obesity (*Sthoulya* or *Medoroga*) as a disorder of the *Medodhatu* (fat tissue) caused by impaired *Agni* (metabolic fire) and *Kapha* predominance. The classical properties of LM herbs include *Lekhana* (scraping or reducing accumulated fat) and *Medohara* (fat-reducing), which are

attributed to bitter, pungent and astringent tastes and light, dry qualities. The present findings offer molecular underpinnings for these ancient descriptors.

The thermogenic activation via ADRB3 and UCP1 can be interpreted as an enhancement of *Agni*, promoting the transformation of accumulated *Medodhatu* into heat energy, a process consistent with *Lekhana*. Meanwhile, modulation of monoamine pathways that influence satiety mirrors the Ayurvedic concept of balancing *Vata* and *Kapha* to reduce cravings and lethargy. For example, increased dopaminergic tone may counteract the sluggishness associated with *Kapha* dominance, while elevated serotonergic signalling could harmonise Vata, reducing anxiety-driven eating.

Furthermore, the identification of targets involved in lipid synthesis and cortisol metabolism parallels the Ayurvedic notion of correcting fat tissue dysmetabolism (*Medo Dhatu Dushti*). HSD11B1 inhibition is reminiscent of herbs described as *Kaphahara* (Kapha-pacifying), which aim to reduce viscous, sticky qualities associated with excess adiposity. Altogether, these molecular correlations bridge classical Ayurvedic theory with contemporary pharmacology, providing a rationale for why a combination of LM herbs, many of which have bitter or pungent tastes, may synergistically rectify metabolic imbalances.

Comparative evidence from modern studies

Many phytochemicals in LM have been individually investigated for anti-obesity effects, offering external validation of the network predictions. Berberine, a key constituent of *Daruharidra* (*Berberis aristata* D.C.), enhances energy expenditure by upregulating UCP1 in brown adipose tissue and improves insulin sensitivity through AMPK activation. [31,32] Curcumin, the principal polyphenol in *Haridra* (*Curcuma longa* L.), reduces adipogenesis, enhances β-oxidation and modulates serotonin and dopamine levels.[33] *Vacha* (*Acorus calamus* L.) contains β-asarone and acorin, which display MAO-inhibitory activity and neuroprotective effects.[34] Network analysis indicates that these compounds, when combined, may exert additive or synergistic effects on ADRB3, MAOA and SLC6A3.

Clinical trials lend support: Berberine supplementation reduces body mass index and improves lipid profiles in obese individuals, [35] while curcumin is associated with reduced waist circumference and inflammatory markers.[36] The polyherbal combination used in LM may therefore harness complementary mechanisms. For instance, while berberine primarily improves metabolic parameters and thermogenesis, curcumin may modulate inflammatory pathways and neurotransmitter balance. The inclusion of *Picrorhiza kurroa* and *Holoptelea integrifolia* adds hepatoprotective and anti-inflammatory phytochemicals, potentially mitigating adverse effects. [37,38] [39]Such overlap between modern evidence and the in-silico predictions strengthens confidence that LM's traditional use has a tangible pharmacological basis.

Limitations and future directions

Network pharmacology relies on predicted interactions; although stringent criteria were used (bioavailability score ≥ 0.55, target probability ≥ 0.4), and current databases were consulted, predictions

require experimental confirmation. Moreover, the analysis focused on human targets; interactions relevant to gut microbiota or non-canonical receptors may have been overlooked. Third, synergy was inferred qualitatively by mapping multiple compounds to shared hubs and pathways; quantitative synergy models (e.g., combination indices) were not applied. Fourth, LC-MS/MS identified 44 compounds, but the extract likely contains additional metabolites that were below detection limits or present as conjugates. Moreover, in vivo bioavailability and metabolism of these phytoconstituents may differ from SwissADME predictions.

Future work should integrate experimental validation at multiple levels. In vitro assays could confirm binding of LM compounds to ADRB3, MAOA and other hub proteins, while cellular models could assess effects on thermogenesis, lipid accumulation and neurotransmitter release. Metabolomic and pharmacokinetic studies are needed to determine which constituents reach systemic circulation. Animal studies could evaluate the formulation's impact on body weight, energy expenditure, mood, and potential toxicity. Additionally, exploring gut–brain axis modulation by LM may reveal probiotic or microbiota-dependent mechanisms. Finally, clinical trials assessing standardised LM extracts would be the ultimate step to confirm efficacy and safety in human obesity.

Conclusion

This study bridges ancient Ayurvedic wisdom with modern systems pharmacology, elucidating how the classical *LekhaniyaMahakashaya* formulation may combat obesity through multi-target modulation. By highlighting adrenergic thermogenesis, monoamine-mediated satiety and improved metabolic homeostasis, the findings provide a mechanistic context for traditional concepts of *Lekhana* and *Medohara*. The formulation's polyherbal nature appears to produce a network synergy that single compounds alone might not achieve. Nonetheless, these results are predictive; rigorous experimental validation is essential. From a translational perspective, LM exemplifies how traditional knowledge can inform modern drug discovery by focusing on poly-component, multi-target therapies.

Declarations

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Table

Table 1 is available in the Supplementary Files section.

Figures



Figure 1

Formulation Composition of *Lekhaniya Mahakasaya*

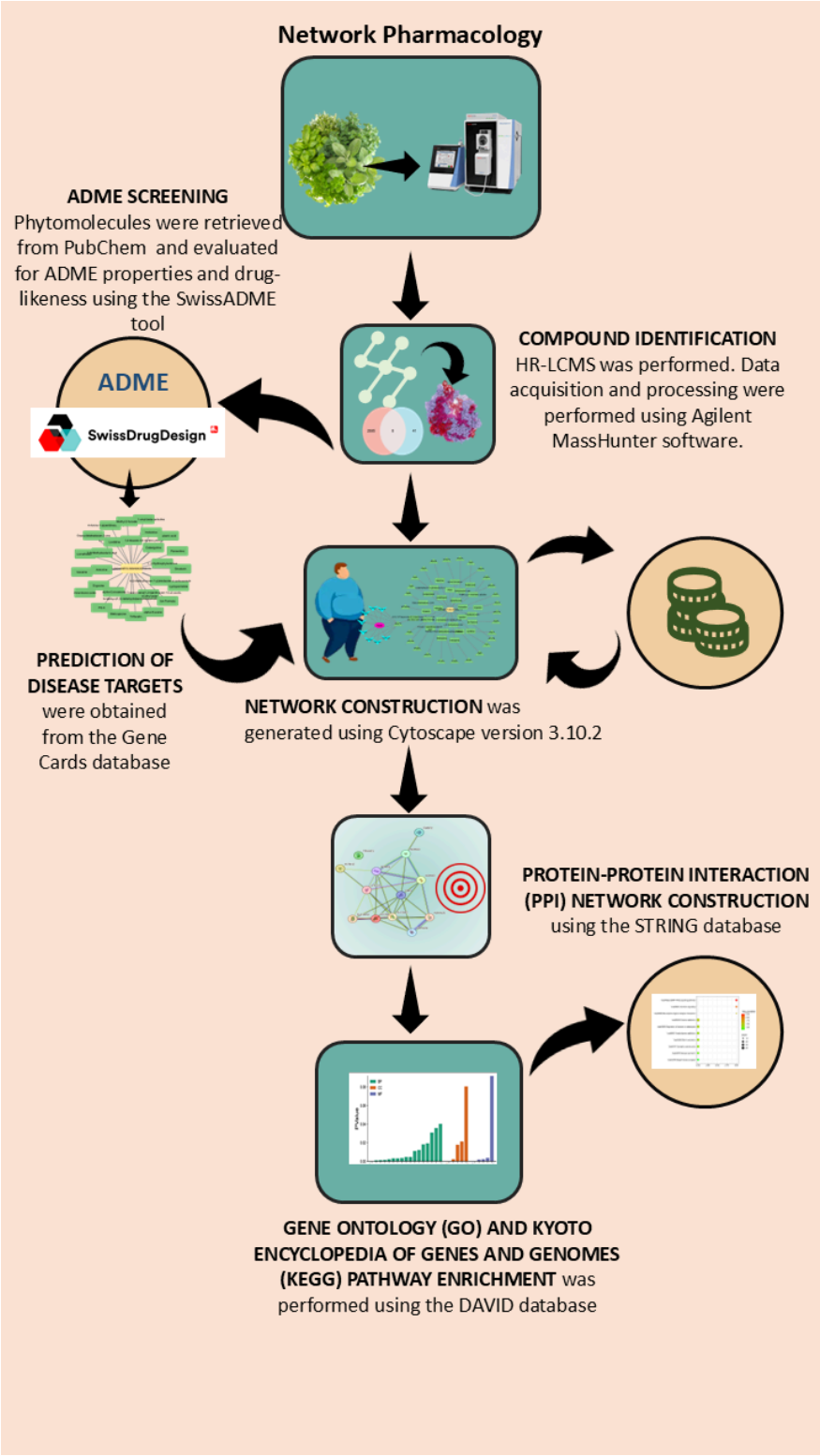


Figure 2

Methodology in the Insilco Network Pharmacology Analysis of LM

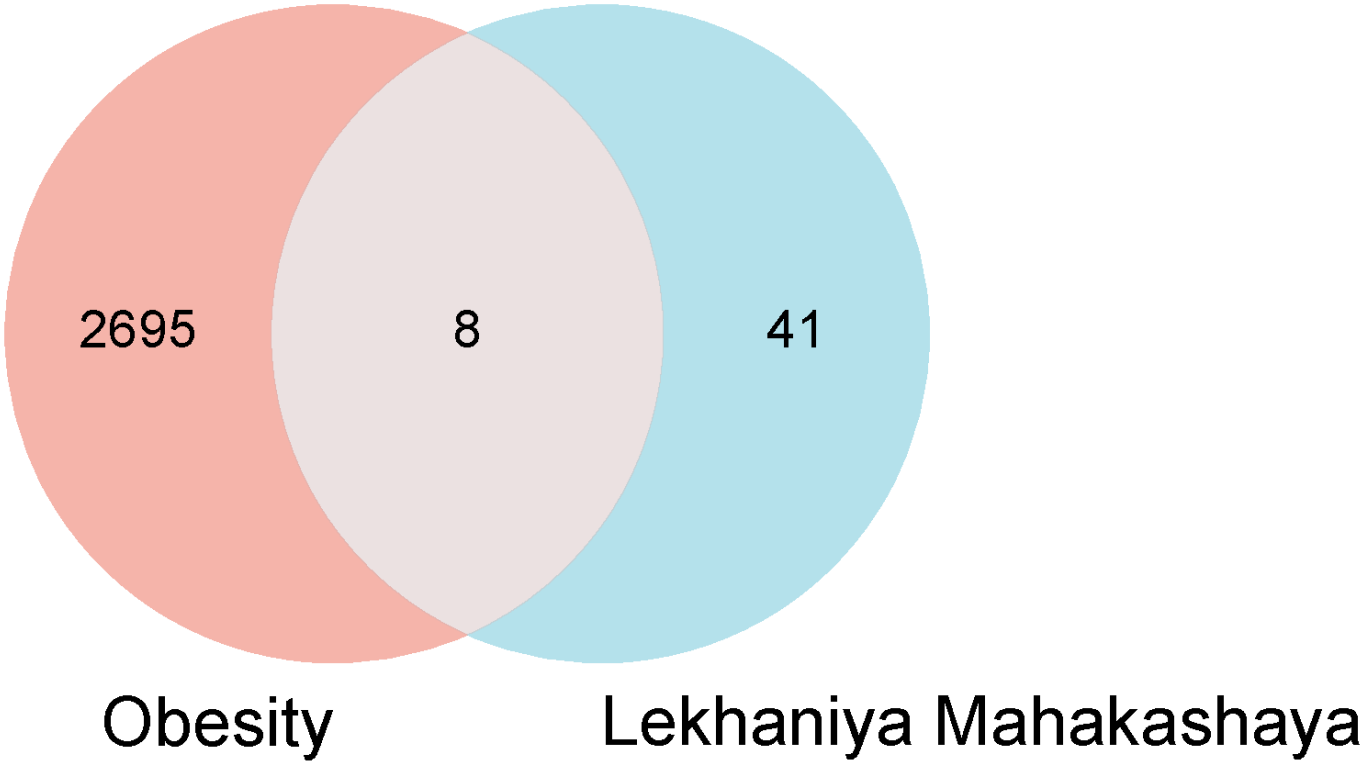


Figure 3

Overlap of predicted targets of *Lekhaniya Mahakashaya* (LM) and obesity-associated genes

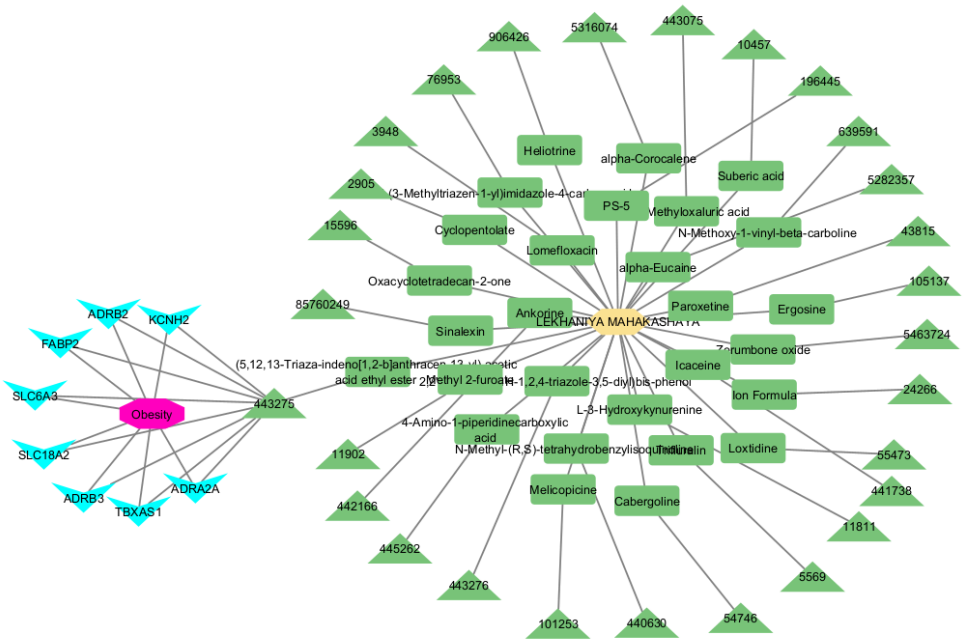


Figure 4

Phytochemical–Target–Disease Interaction Network of *Lekhaniya Mahakashaya* (LM)

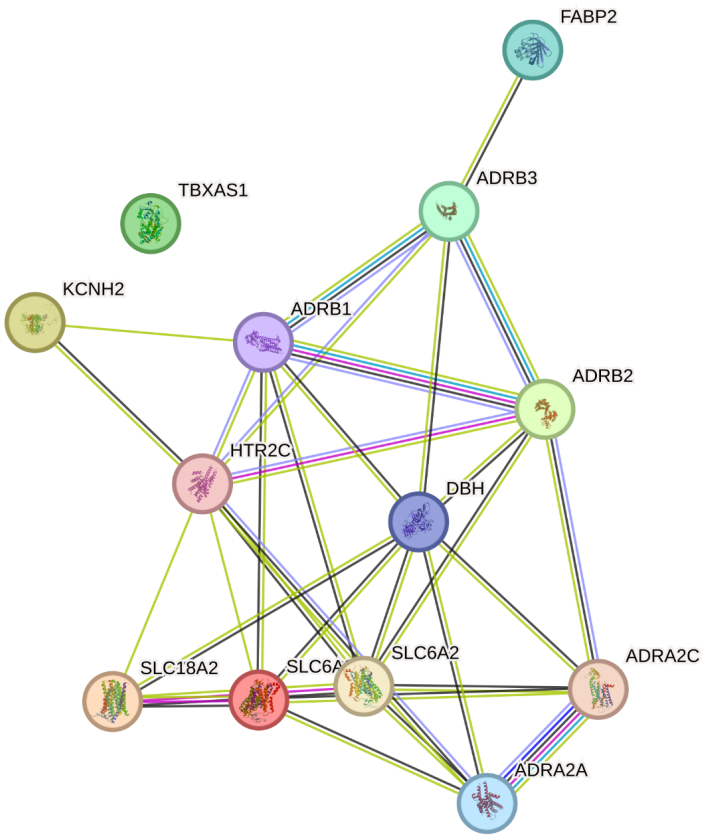


Figure 5

Protein–protein interaction network showing eight overlapping targets between LM and obesity-related genes

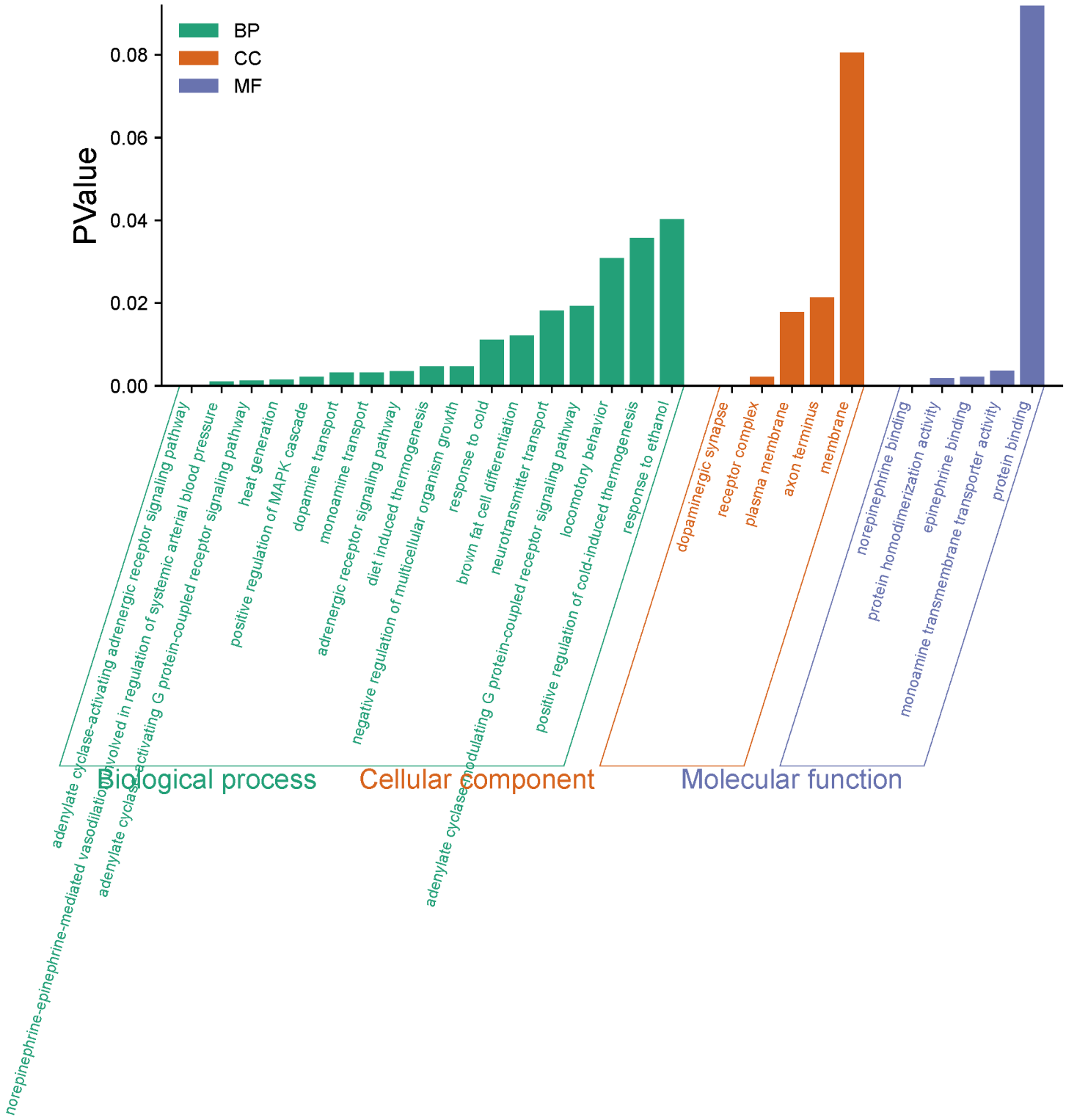


Figure 6

Gene Ontology (GO) enrichment analysis of overlapping targets between *Lekhaniya Mahakashaya* and obesity

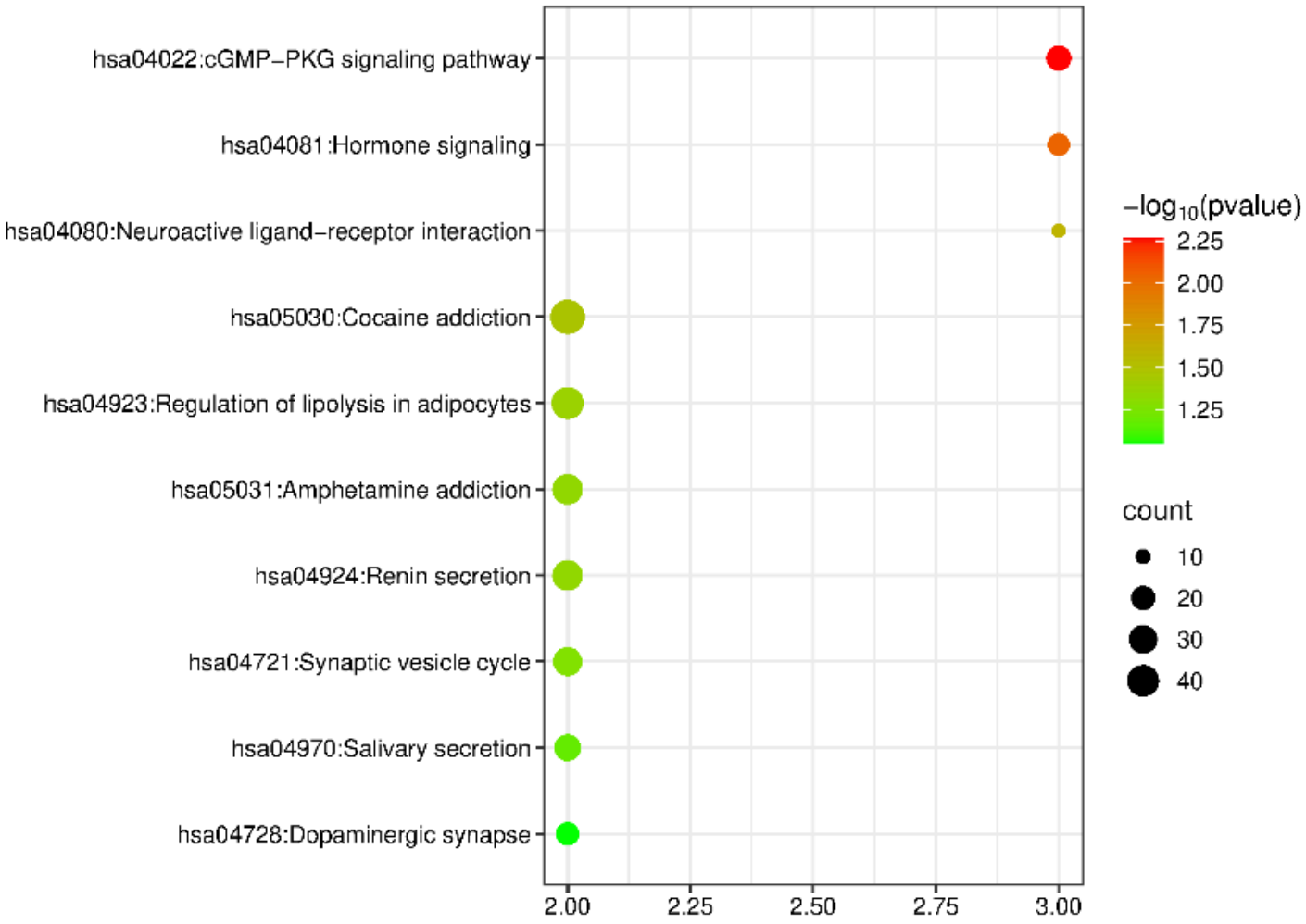


Figure 7

KEGG pathway enrichment analysis of overlapping targets between *Lekhaniya Mahakashaya* and obesity

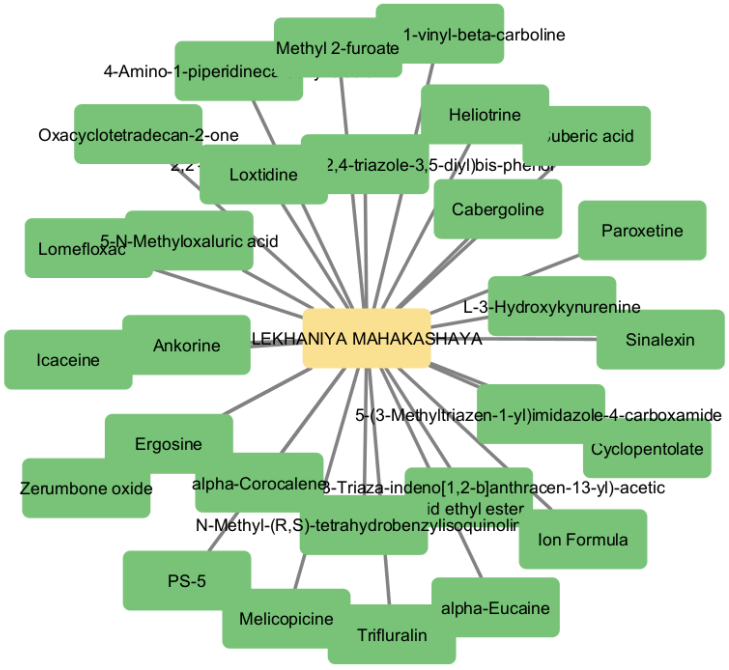


Figure 8

Network of ADME-qualified phytochemicals identified in *Lekhaniya Mahakashaya* (LM) by HR-LC-MS/MS

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- MoleculeDone.docx
- Table1.docx
- TargetMoleculesdone.docx