RESEARCH PAPER

Synergistic Wound-Healing Potential of Green Tea and Rosemary Bioactives: Insights from Network Pharmacology and Molecular Docking

Jatin Pachera1, Akshay Verma[1], Balwan Singh[1] and Shardendu Kumar Mishra[1*]

[1]*KIET School of Pharmacy, Krishna Institute of Engineering & Technology (KIET), Ghaziabad, Delhi-NCR, Uttar Pradesh, India*

[*]***Corresponding Author:*** *Dr. Shardendu Kumar Mishra, Associate Professor, Department of Pharmacology, KIET School of Pharmacy, Krishna Institute of Engineering & Technology (KIET), Ghaziabad, Delhi-NCR, Uttar Pradesh, India*

Email: shubham.dr@gmail.com

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ABSTRACT

Wound healing represents a highly coordinated biological sequence that encompasses inflammation, cellular proliferation, and tissue remodeling, all working together to repair and restore the integrity of damaged skin. Disturbances in immune response, infection, and oxidative stress delay healing. Plant-derived phytochemicals such as those from green tea and rosemary may enhance healing by targeting multiple molecular pathways simultaneously. Active compounds of polyherbal preparation were collected from literature databases and screened for ADMET and drug-likeness using SwissADME. SwissTargetPrediction helped forecast potential targets for these compounds, while GeneCards supplied the wound healing-related genes. Venny 2.1 then flagged the overlapping targets between drug effects and disease genes. From there, we built a PPI network in STRING and dug into it with Cytoscape to spot hub genes, followed by GO and KEGG analysis to map out key functions and pathways. Finally, molecular docking was carried out between key phytochemicals and core wound-healing targets to evaluate binding affinity. 8 major bioactive compounds were identified from green tea and rosemary. Target prediction yielded 481 potential drug targets, while 7,101 wound-healing–related genes were retrieved from GeneCards. Intersecting analysis identified 262 common targets. PPI network analysis revealed 10 hub genes (IL6, HIF1A, EGFR, TP53, TNF, SRC, ESR1, CASP3, PPARG, BCL2). GO enrichment indicated involvement in inflammation, cell proliferation, and re-epithelialization. KEGG analysis highlighted the MAPK signaling pathway as the most prominent pathway related to wound healing. Molecular docking studies revealed robust binding affinities for key compounds-including rosmarinic acid, ursolic acid, carnosic acid, carnosol, EGCG, ECG, catechin and EGC-with pivotal targets like IL-6, TP53, EGFR, TNF and SRC, therby bolstering their promise in promoting wound healing. The findings from this study lay a solid groundwork for deeper exploration into the wound-healing compounds and pathways in green tea and rosemary, while offering a scientific rationale for developing novel therapeutics agents derived from their bioactive phytochemicals.

Keywords: Wound-healing; Rosmarinus, Camellia; Network pharmacology; Molecular docking

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INTRODUCTION

Wound healing stands out as an intricate, tightly controlled biological cascade essential for rebuilding tissue structure and preserving bodily balance following injury. Far from being a series of standalone steps, it unfolds across interconnected phases-hemostasis, inflammation, proliferation and remodeling, as depicted in **Figure 1**- that collectively drive successful repair. The pace and success of this process hinge on local wound-site dynamics as well as broader systemic influences. Central to it all are processes like inflammatory cascades, fibroblast mobilization, and extracellular matrix reorganization. Vital cellular activities, including migration, new blood vessel formation, and re-epithelialization, prove indispensable for genuine tissue regeneration.[1]

**Author for Correspondence: shubham.dr@gmail.com*

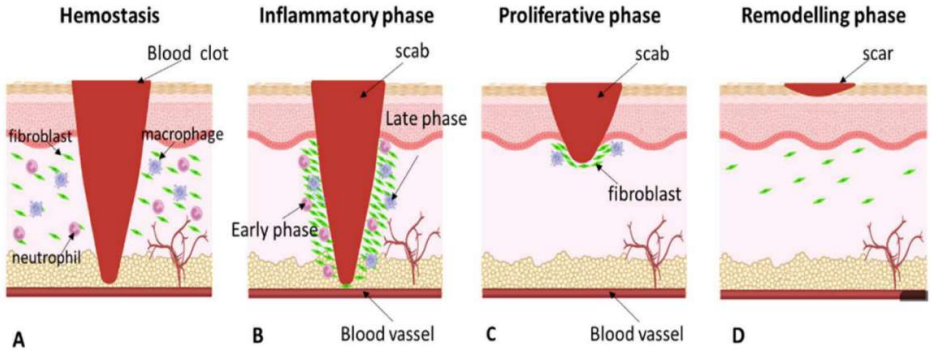


Figure 1: Sequential stages of wound healing.[2]

Individual genetic differences can significantly affect the production of proteins and growth factors required for normal wound repair. Moreover, systemic disorders such as diabetes, vascular insufficiency, and chronic infections are known to delay healing and may result in the progression of chronic and non-healing wounds.[3]

In many cases, impaired wound healing is associated with prolonged or dysregulated inflammation, which can ultimately lead to pathological conditions, including excessive scar formation or fibrosis.[4]

This study leveraged network pharmacology alongside molecular docking to identify bioactive phytochemicals, their molecular targets, and key pathways underlying the wound healing potential of our polyherbal preparation (**Figure 2**).

MATERIAL AND METHODS

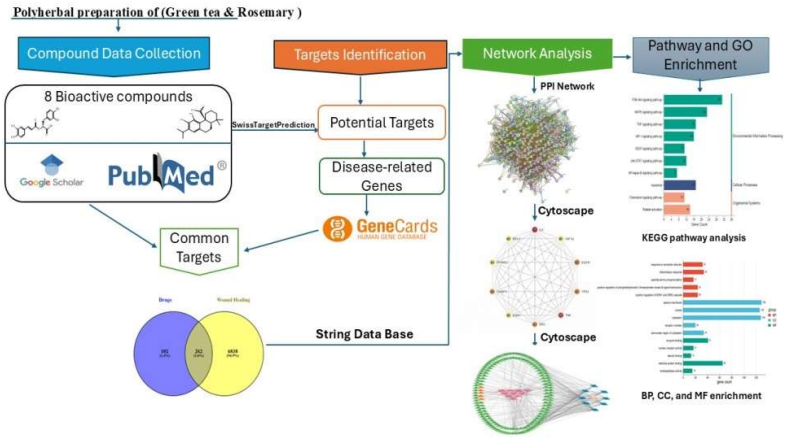
Identification and Collection of Bioactive Compounds

Step 1:

Bioactive compounds of the studied plants were retrieved from PubMed, Google Scholar. SwissADME (https://www.swissadme.ch/) web server helped us forecast the ADMET profiles (absorption, distribution, metabolism, excretion, and toxicity) for all compounds. This platform also evaluated drug-likeness by assessing the Lipinski rules, as well as the bioavailability score.[5]

Prediction of targets for active phytoconstituents in polyherbal preparation

Likely protein targets linked to the active constituents present in polyherbal preparation composed of two medicinal plants, green tea *(Camellia sinensis)* and rosemary *(Rosmarinus officinalis)*, were identified using the SwissTargetPrediction online platform, considering targets with a probability score greater than zero. Target prediction analysis was performed via the SwissTargetPrediction platform (https://www.swisstargetprediction.ch/, retrieved on November 3, 2025).[6]



Step 2:

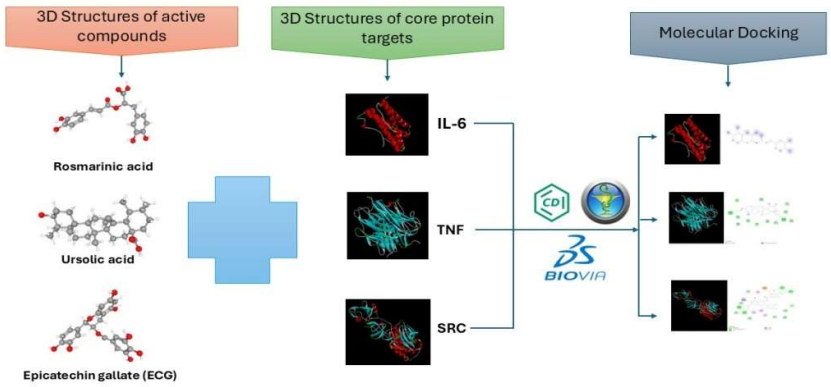


Figure 2: Schematic overview of the study design

Screening of disease-related genes

In this study, Wound healing-associated genes were collected using the GeneCard® Human Gene Database on November 3,2025 (https://www.genecards.org/).The search was performed using the terms "Wound Healing". The retrieved genes were subsequently used for further analysis.[7]

Screening of Interlinked targets

Using Venny 2.1 web software (https://bioinfogp.cnb.csic.es/tools/venny/, retrieved November 3, 2025), we identified proteins serving as compound targets that also stem from wound healing-related genes. The common entities were evaluated to be prospective targets of polyherbal preparation against wound healing.

Protein-Protein network evaluation

Protein-Protein binding analysis of the possible targets of polyherbal preparation against wound healing was carried out via the STRING database (Version 12.0; https://strings-db.org/:) retrieved on November 4, 2025).[8] Interactions with confidence scores greater than 0.4 were considered significant, and the analysis was restricted to Human organism as a result PPI network was subsequently imported into Network graphing tool {Cytoscape}(Version 3.7.2 accessed on November 4, 2025) for visualization and further analysis.[9]

Compound-target network construction

Investigation and exploration of PPI network was done with the help of Cytoscape software (https://cytoscape.org/). Cytohubba, an application within the Cytoscape software, was used to retrieve top 10 nodal targets based on degree value (topology parameter).[10] These nodal targets will be used for further analysis.

Enrichment study

Functional GO annotation enrichment alongside KEGG metabolic pathway analysis was carried out via the DAVID database (Version 7.8; https://david.ncifcrf.gov/) for gene categorization, graphical representation, and unified analysis. Gene ontology (GO) terms fell into the usual three buckets: Molecular function (MF), Cellular components (CC), and Biological process (BP). That obtained details were further uploaded into the SR PLOT (https://www.bioinformatics.com.cn/en) online platform to generate bubble plots illustrating the enriched biological processes and pathways. Statistical relevance was evaluated using the Hypergeometric distribution test. To account for multiple comparisons, p-values were corrected using the (BH) Benjamini Hochberg procedure for FDR (False positive percentage) correction, where adjusted $p< 0.05$ indicated significance.[11]

In silico molecular docking analysis

Molecular docking serves as a key computational technique for forecasting how small-molecule ligands might orient themselves and bind to target receptor proteins. Here, we chose docking targets from the central nodes in wound healing-related pathways, pinpointed earlier via KEGG pathway enrichment analysis. The RCSB Protein Data Bank (https://www.rcsb.org/) was used to acquire the 3D crystal structures of the identified target proteins in PDB format.

Protein preparation, including structure refinement and optimization, was carried out using Discovery Studio Visualizer (BIOVIA) (https://www..3ds.com/products/biovia/discovery-studio). The Canonical SMILES of the active compounds were utilized to generate their two-dimensional (2D) chemical structures prior to docking. We performed molecular docking of the prepared ligands against selected target proteins using PyRx tool (https://pyrx.sourceforge.oi/). Following docking, the Protein-ligand complexes were visualized and analyzed to assess binding interactions using BIOVIA Discovery Studio Visualizer.[12-14]

RESULTS

Active compounds of polyherbal preparation

In the first step, 76 phytoconstituents associated with the polyherbal preparation were gathered through an extensive literature survey from multiple scientific databases including- ScienceDirect, Scopus, PubMed and other related sources. Further active compound screening was carried out using SwissADME, where oral bioavailability (OB ≥ 0.55) and Lipinski rule (drug likeness)- 0 Violation criteria were applied. Based on these ADME parameters, 20 compounds were initially obtained. Subsequently, toxicity-based screening was performed by using (Protox 3.0 software), presented in **Table 1** and resulted in the identification of 8 active compounds **(Figure- 3-i),** as illustrated in **Table 2.**

Predicted targets of active Phytoconstituents

Target prediction based on ligand similarity was carried out for the polyherbal constituents using SwissTargetPrediction (https://www.swisstargetprediction.ch/) web tool. This analysis initially yielded 499 possible targets with a probability score greater than zero. After removing duplicate entries, a total of 481 unique predicted targets were retained for subsequent analyses **(Figure 3-ii)**

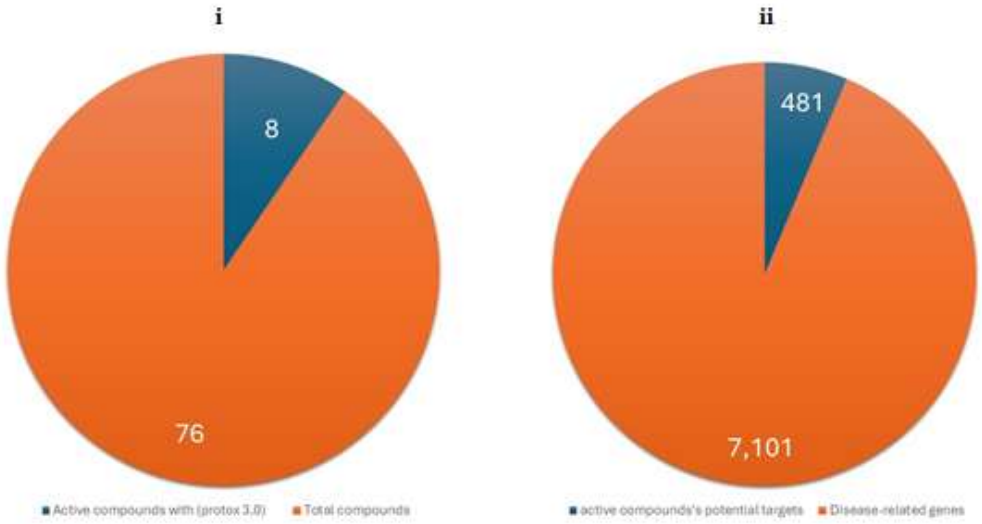
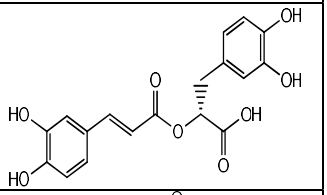
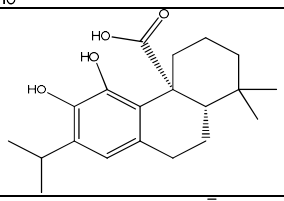
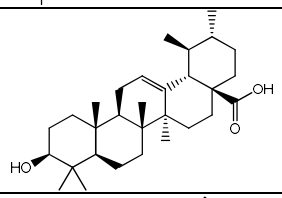
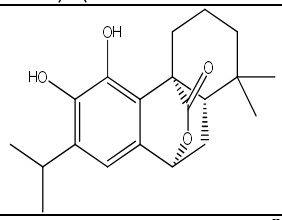
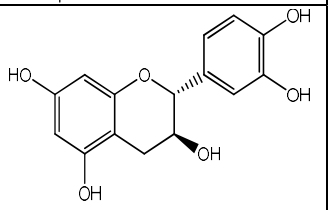
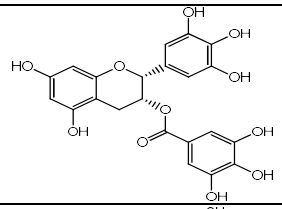
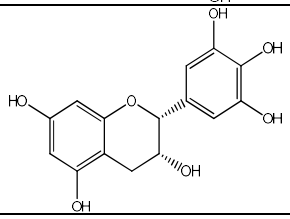


Figure 3: (i) Overall compounds versus active compounds in polyherbal formulation. **(ii).** Potential targets of active compounds alongside wound healing-related genes.

Table 1: Active compounds selected through toxicity screening

Plant Name: *Rosmarinus officinalis*		
Active Phytoconstituents	**Lipinski rule**	**Toxicity** (*Hepatotoxicity, Nephrotoxicity, Cytotoxicity, Cardiotoxicity, Neurotoxicity, Respiratory toxicity, Carcinogenicity*)
Rosmarinic acid	0 violation	Inactive
Carnosic acid	0 violation	Inactive
Ursolic acid	0 violation	Inactive
Carnosol	0 violation	Inactive
Plant Name: *Camellia sinensis*		
Active Phytoconstituents	**Lipinski rule**	**Toxicity** (*Hepatotoxicity, Nephrotoxicity, Cytotoxicity, Cardiotoxicity, Neurotoxicity, Respiratory toxicity, Carcinogenicity*)
Catechin	0 violation	Inactive
Epigallocatechin (EGC)	0 violation	Inactive
Epigallocatechin gallate (EGCG)	0 violation	Inactive
Epicatechin gallate (ECG)	0 violation	Inactive

Table 2: Active compounds of polyherbal preparation

S. No.	Compound name	CID	MF	OB (%)	DL	Structure of compounds
1.	Rosmarinic acid	5281792	$C_{18}H_{16}O_8$	1.38	0.35	
2.	Carnosic acid	65126	$C_{20}H_{28}O_4$	0.55	0.55	
3.	Ursolic acid	64945	$C_{30}H_{48}O_3$	16.77	0.75	
4.	Carnosol	442009	$C_{20}H_{26}O_4$	14.96	0.43	
5.	Catechin	9064	$C_{15}H_{14}O_6$	54.83	0.24	
6.	Epigallocatechin gallate (EGCG)	65064	$C_{22}H_{18}O_{11}$	55.09	0.77	
7.	Epigallocatechin (EGC)	72277	$C_{15}H_{14}O_7$	24.18	0.27	

8.	Epicatechin gallate (ECG)	107905	$C_{22}H_{18}O_{10}$	3.01	0.75	

Disease-related genes

Searching GeneCards (https://www.genecards.org/) with "wound healing" as the Keyword uncovered 7,101 related genes were identified (**Figure 3-ii**).

Venn diagram showing overlaps between wound healing gene products and compounds targets

Using the Venny 2.1.0 web tool, we pinpointed overlapping targets between wound healing gene products and compounds hits. These overlaps appear in **Figure 4**. Overall, 262 shared targets emerged as promising candidates for the polyherbal preparation's effects on wound healing.

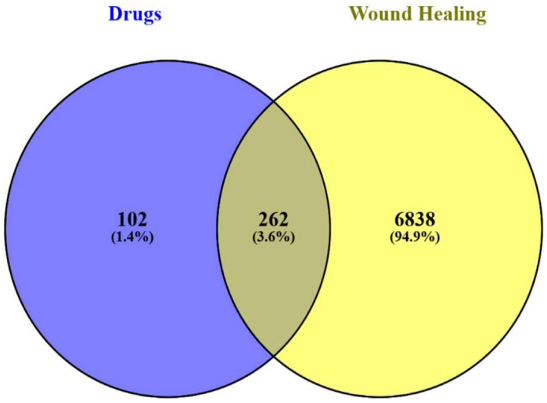


Figure 4: Venn diagram illustrating overlaps between wound healing genes and targeted proteins

Study of protein-protein interaction (PPI) network

We built the PPI network for those 262 potential targets of green tea and rosemary in wound healing by loading them into the STRING database (version 12.0). Analysis ran under strict settings-limited to Homo sapiens with a confidence cutoff of ≥0.9-to guarantee solid physical and functional links. Cytoscape 3.7.2 handled visualization and analysis, producing a network of 262 protein nodes linked by 3,789 edges (**figure 5-i**). (**figure 5-ii)** uses node size and color to represent degree values, highlighting connectivity strength. By applying the CytoHubba tool, five hubs' proteins- **EGFR, TP53, TNF, IL-6** and **SRC**-were identified based on their topological significance, as illustrated in Figure 6b. These hub genes, detailed in **Table 3** are highly influential within the network and may serve as critical therapeutic targets for wound healing.

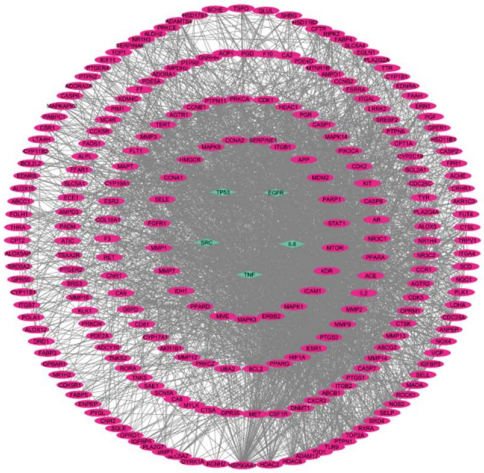
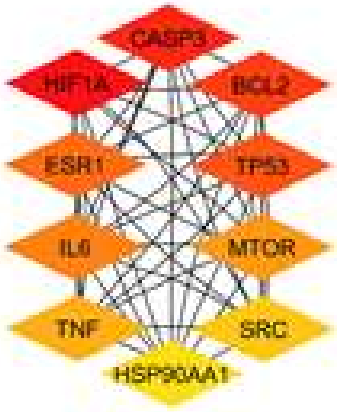
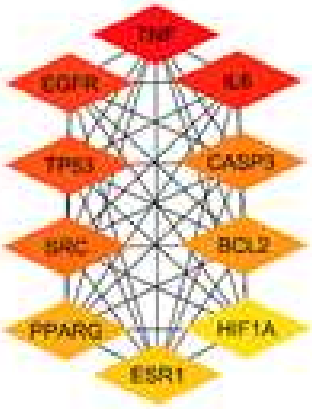


Figure 5: (i) PPI Network of Active Compounds of Polyherbal Preparation against wound healing

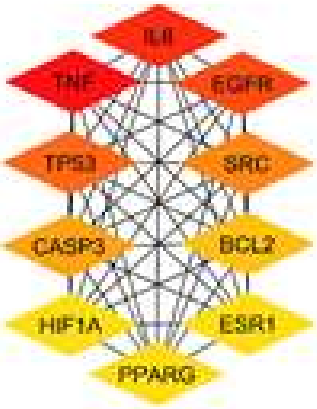
a. MCC



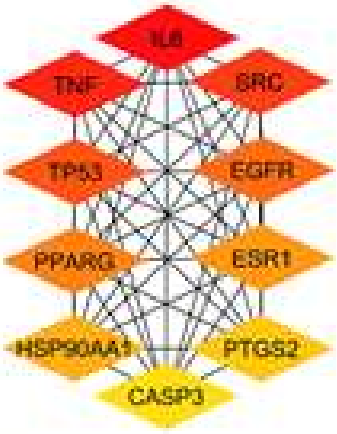
b. Closeness



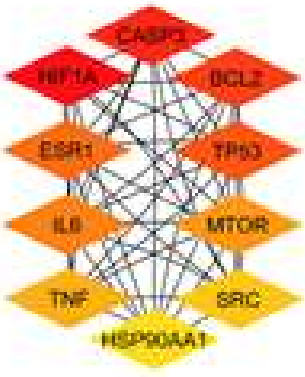
c. Degree



d. Betweenness



e. MNC



f. Venn diagram of 5 CytoHubba algorithms

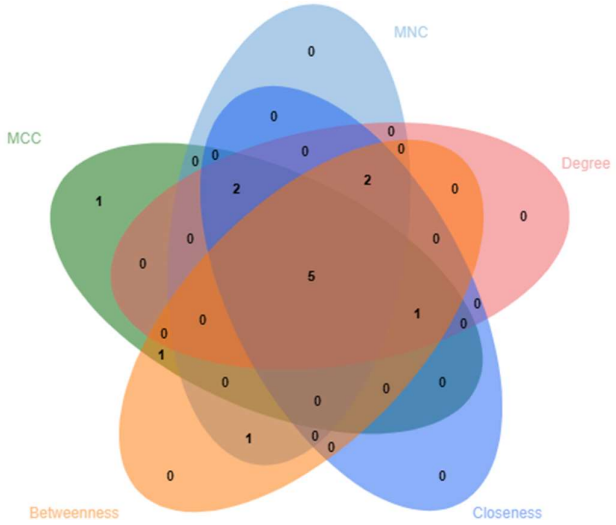


Figure 5 (ii): Presents a network diagram showcasing the main targets of polyherbal preparation in wound healing, using 5 CytoHubba algorithms: (a) MCC, (b) Closeness, (c) Degree, (d) Betweenness, & (e) MNC. Additionally, (f) includes a Venn figure displaying the 5 common hub hub proteins identifies.

Table 3: Topological properties of the 5 potential hub targets.

(5 Hub) proteins	Degree	MCC	MNC	Closeness	Betweenness
TNF	151	9.223E+13	151	204.5	4919.9562
IL-6	148	9.223E+13	148	203.0	5147.44984
EGFR	129	9.223E+13	129	193.5	3285.489
SRC	118	9.223E+13	118	188.0	4007.96482
TP53	124	9.223E+13	124	191.0	3394.90655

Network of active compounds and targets of polyherbal preparation against wound healing

Using Cytoscape 3.7.2, we generated a network to map out the interaction among the eight active compounds (identified in the "Active compounds of green tea and rosemary" section) and the 262 targets of polyherbal preparation against wound healing (identified in the "Analysis of (PPI)" section) (**Figure 6**). Within this network, every edge captures a predicted interaction between an active compound and a wound healing target from the polyherbal preparation.

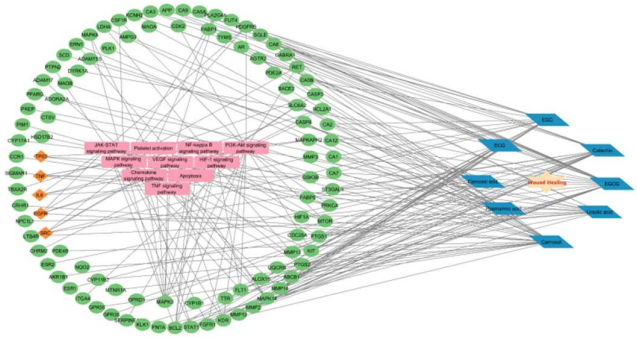


Figure 6: Network depicting eight key active compounds alongside their corresponding targets in the polyherbal preparation against wound healing

Enrichment analysis highlighting key pathways linked to central nodal targets

Overall, 778 BP, 107 CC and 278 MF was obtained from GO analysis of nodal targets which helps in exploration of biological mechanism of active constituents of polyherbal preparation against wound healing. Bubble plot illustrates the top 5 terms in biological processes (BP), cellular components (CC), and molecular functions (MF) **(Figure**

7-i). Kyoto Encyclopedia of Genes and Genomes analysis provides a total of 154 pathways including various pathways associated with wound healing. The obtained data on pathway enrichment suggests that the polyherbal preparation can potentially target wound healing as the pathways are associated with the hemostasis, Re-Epithelialization, inflammation, proliferation and other pathways. Top 10 pathways were represented as horizontal bar chart **(Figure 7-ii)**. Prominent pathways involved in wound healing mainly include the MAPK signalling pathway which were linked with nodal targets (TNF, EGFR, TP53). Docking analysis was done on these proteins to explore their role and mechanism in treatment of wound healing.

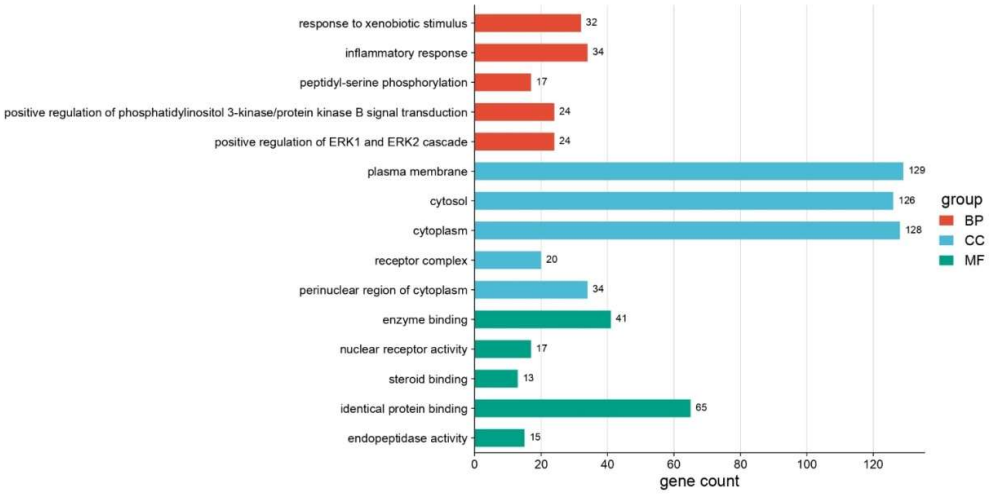


Figure 7-i: BP, CC, MF enrichment

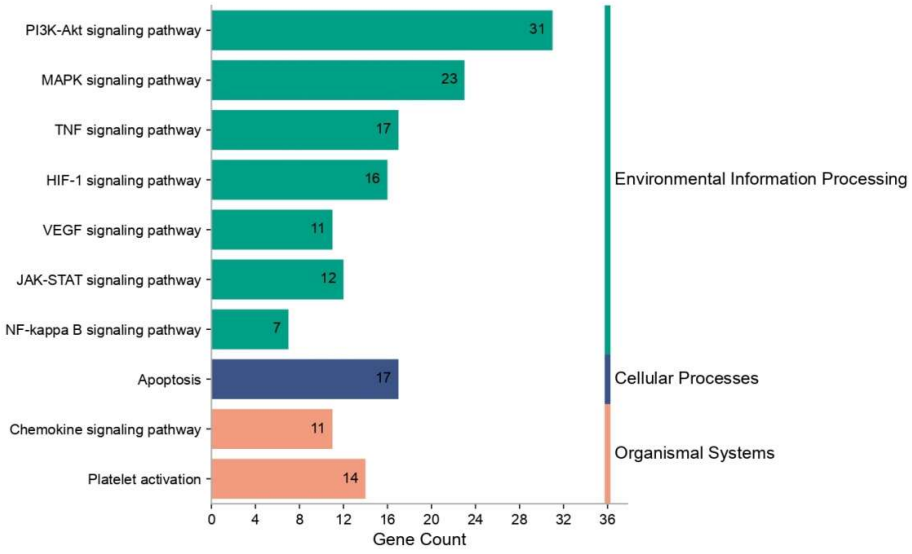


Figure 7-ii: KEGG pathway analysis

Molecular docking

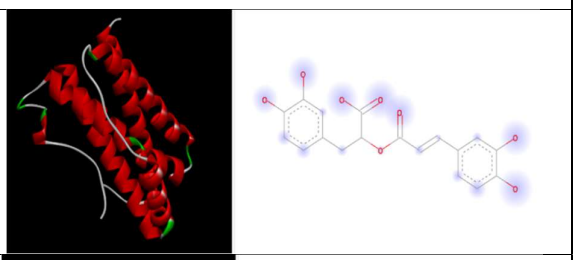
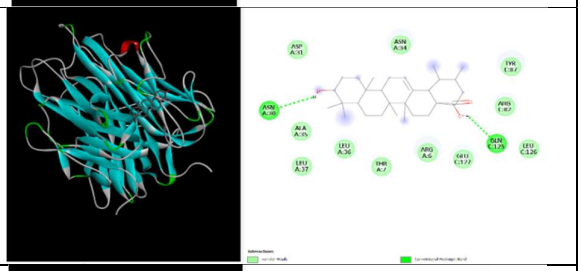
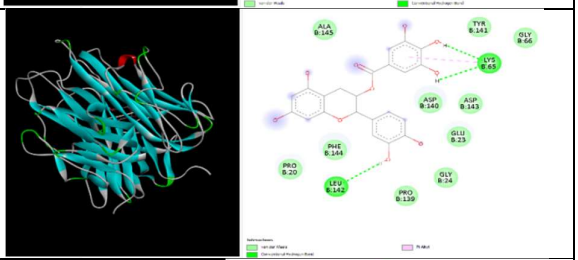
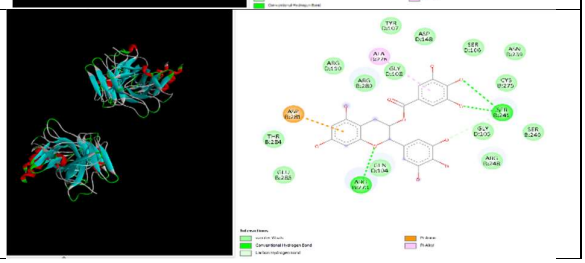
We carried out molecular docking between the key active compounds from polyherbal preparation and the core targets of wound healing (IL-6, TNF, TP53, EGFR, SRC). The docking results appear summarized in **Table 4** and depicted visually in **Table 5**. The strength of interaction between each compound and target protein is expressed as the binding energy score; A lower binding energy generally suggests that the ligand binds more strongly and forms a more stable complex with the receptor.[15]

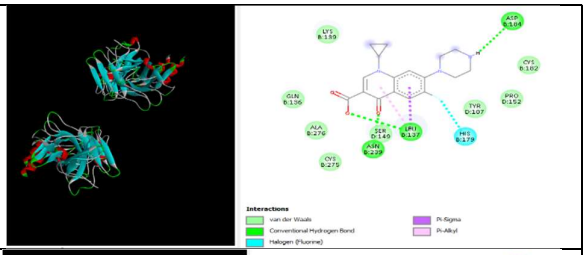
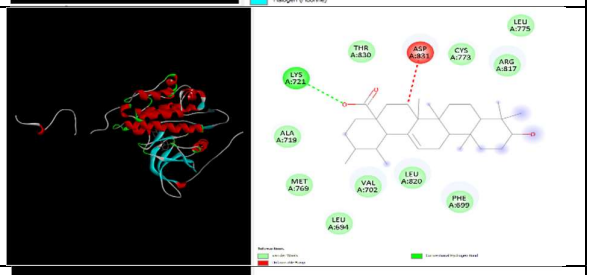
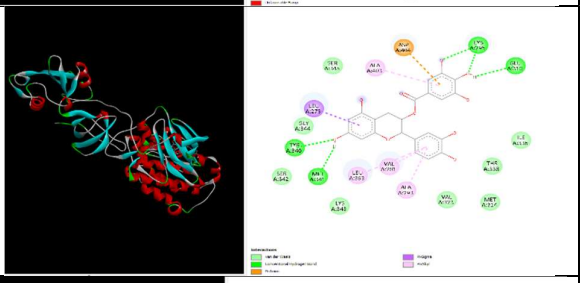
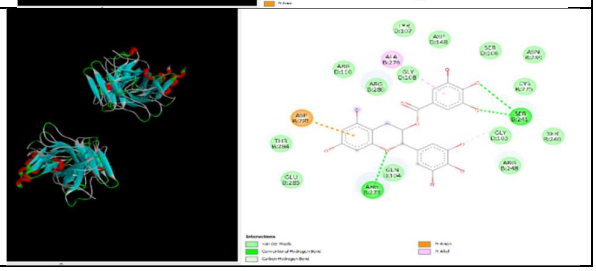
The docking analysis showed that all major active compounds of polyherbal preparation were able to bind with **IL-6, TNF, TP53, EGFR,** and **SRC.** Our results suggest that the main compounds in this polyherbal formulation are likely to promote wound healing through direct interactions with those pivotal targets. What's more, the docking outcomes aligned closely with network pharmacology forecasts, which reinforces the robustness of our network-based methodology.

Table 4: Molecular docking analysis of key bioactive compounds from the polyherbal preparation against central wound healing targets.

Molecular Docking		Binding energy (Kcal/mol)				
Compound name	Gene Name (PDB ID)	IL-6 (1ALU)	TNF (1A8M)	TP53 (2OCJ)	EGFR (1M17)	SRC (1Y57)
Rosmarinic acid		-7.6	-6.6	-6.8	-7.6	-7.9
Carnosic acid		-6.6	-6.2	-6.6	-7.8	-7.6
Carnosol		-7	-7.1	-7.1	-8	-7.7
Ursolic acid		-7.2	-7.6	-6.7	-9.1	-8.5
Catechin		-6.5	-6.2	-6.7	-7.7	-8
Epigallocatechin (EGC)		-6.3	-6.7	-7.2	-7.8	-7.4
Epicatechin gallate (ECG)		-7	-7.4	-7.8	-8.9	-8.9
Epigallocatechin gallate (EGCG)		-7	-7.3	-8.1	-8.4	-8.5
Povidone-iodine		-4	-4.2	-4.6	-4.1	-4.5
Nitrofurazone		-5.4	-6.9	-6.2	-6.1	-5.9
Neomycin B		-5.8	-6.4	-7	-6.9	-7
Ciprofloxacin		-6.3	-6.9	-8	-8.1	-8.2

Table 5: Phytoconstituents exhibiting stronger binding affinity.

Potential Target Name	Lead Compound	2D/3D Figure of Interaction
IL-6	Rosmarinic acid	
TNF	Ursolic acid	
TNF	Epicatechin gallate (ECG)	
TP53	Epigallocatechin gallate (EGCG)	

TP53	Ciprofloxacin	
EGFR	Ursolic acid	
SRC	Epicatechin gallate (ECG)	
TP53	Epigallocatechin gallate (EGCG)	

DISCUSSION

Wound healing unfolds as a dynamic, precisely orchestrated biological process crucial for reinstating normal tissue architecture and functionality.[16] In the present work, a computer-based modeling strategy has been used to assess how a selected phytochemical may influence the time required for healing and its overall effectiveness, by evaluating its interaction with major receptors associated with the wound-healing pathway. A wound amounts to a disruption in the tissue's normal anatomical and physiological continuity, often caused by accidental injury, surgery, or underlying disease conditions.[17] Wounds typically get classified by their origins, falling into acute types like cuts and burns, or chronic ones such as pressure scores and diabetic foot ulcers.[18,19] The healing cascade involves several important cellular contributors, including fibroblasts, keratinocytes, and macrophages, along with Growth factors like Epidermal growth factor receptor (EGFR), transforming growth factor- β (TGF- β) and platelet-derived growth factor (PDGF), all play key roles in driving cell proliferation, angiogenesis, and tissue regeneration.[20, 21] Previous studies indicate that both synthetic drugs and plant-derived compounds can support wound repair through mechanisms including modulation, inhibition, or suppression of specific molecular targets .[22, 23] Therefore, the present study focuses on existing scientific evidence supporting berberine as a promising wound-healing agent and evaluates it through in silico approaches.

In the present work, the wound-healing potential of polyherbal preparation was evaluated using a network-pharmacology and molecular-docking approach. Several phytoconstituents demonstrated strong binding interactions with proteins associated with the MAPK signaling pathway, suggesting that they may serve as promising therapeutic candidates for the management of wound healing. From a total of 76 initially collected compounds, eight bioactive molecules were screened on the basis of oral bioavailability, drug-likeness, and toxicity assessment using the Protox 3.0 platform. Target prediction for these compounds yielded 481 potential protein targets after removal of duplicates. In parallel, 7,101 wound healing-related genes were pulled from the GeneCards database, revealing 262 overlapping targets between disease and compounds were identified using Venny.

We fed these overlapping targets into the STRING database to build a protein-protein interaction (PPI) network, which we then visualized in Cytoscape. To pinpoint the most influential nodes, we applied several centrality measures-Maximal Clique Centrality (MCC), Degree, Betweenness centrality and Closeness centrality-yielding the top ten hub genes as prime core targets such as **Degree-** (IL6, EGFR, SRC, BCL2, ESR1, PPARG, HIF1A, CASP3, TP53, TNF), **MCC-** (CASP3, BCL2, TP53, MTOR, SRC, HSP90AA1, TNF, IL6, ESR1, HIF1A), **MNC-** (CASP3, BCL2, TP53, MTOR, SRC, HSP90AA1, TNF, IL6, ESR1, HIF1A), **Closeness-** (TNF, IL6, CASP3, BCL2, HIF1A, ESR1, PPARG, SRC, TP53, EGFR), and **Betweenness-** (IL6, SRC, EGFR, ESR1, PTGS2, CASP3, HSP90AA1, PPARG, TP53, TNF). Furthermore, Building on their strong topological scores and broader network impact, we zeroed in on the top five hub genes-IL-6, EGFR, TP53, SRC and TNF- for molecular docking experiments. These genes likely hold key regulatory sway in wound healing, marking them as prime candidates for therapeutics intervention. DAVID-based functional annotation turned up 1,163 enriched terms spanning biological processes, molecular functions, cellular components, and KEGG pathways. Drilling down into the leading GO terms and pathways highlighted the MAPK signaling route as the standout player in wound healing, especially via targets like TNF, EGFR and TP53.

A pivotal aspect of the mitogen-activated protein kinase (MAPK) signaling pathways in wound healing lies in how its activation-especially along the ERK and p38 arms-ramps up fibroblast function, boots collagen production, and accelerates tissue repair. Treating cells with potassium chloride (KCL) to stimulate potassium channels triggered robust ERK and p38 MAPK activity, which ramped up collagen synthesis and fine-tuned angiogenic factors in human dermal fibroblasts. These changes prove vital for proper wound closure: collagen lays down the structural framework for new tissue, while regulated angiogenesis delivers the necessary oxygen and nutrients to the site. In essence, MAPK signaling bridges ionic cues to the cellular machinery of repair. Taken together, these insights position the MAPK pathway as a prime target for therapies aimed at improving wound healing and regeneration.[24,25,26]

Docking studies backed this up, demonstrating strong binding poses for rosmarinic acid, carnosic acid, ursolic acid, carnosol, catechin, epicatechin gallate (ECG), epigallocatechin gallate (EGCG) and epigallocatechin (EGC) against key wound-healing targets like IL-6, EGFR, TP53, SRC and TNF. All told, our findings point to these phytochemicals driving re-epithelialization, tempering inflammation, and boosting proliferation via MAPK pathways.

CONCLUSION

The MAPK signaling cascade sits at the heart of wound repair phases like re-epithelialization, inflammation management, and proliferative growth. In our work, various phytochemicals showed robust binding to core pathway proteins, hinting at their capacity to steer healing-related cellular events. Docking results reinforced this, suggesting these compounds could improve repair by targeting MAPK-linked proteins. On balance, they emerged as strong contenders for wound-healing application. That said, Further *in vitro* and *in vivo* studies remain essential to confirm efficacy and safety.

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Conflict of Interest

None of the author is having any conflict.

Declaration of Competing Interest

None

Funding Source

None

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