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Research Article

Molecular Docking Analysis of Tridax Procumbens Phytocompounds Targeting Wound Healing Properties

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ABSTRACT

Wound healing is a multicellular and complicated process that strives to restore the skin's barrier function. Different cell types, including keratinocytes, endothelial cells and fibroblasts collaborate for the process to complete. Tridax procumbens is a very important natural plant that is commonly found in the tropical region and has a wide range of pharmacological actions. In the present study the molecular docking was used to identify the activity of four compounds from T. procumbens to the selected receptor. The goal of this work was to anticipate the capacity of T. procumbens compounds to interact with , IL-6. Pyrx was used to run docking simulations for these compounds. Results of this study showed that all the compounds showed interaction with selected target proteins. Among the compounds, Cynaroside, Quercetin, Linoleic, Linolenic, Palmitoleic acid, Campesterol, 9-Heptadecanone showed excellent binding and hydrogen bond interaction with all the selected target proteins. The results showed that T. procumbens compounds are effective anti-inflammatory agents. However, further research is required to validate the action of these molecules.

INTRODUCTION

1.1 Drug Discovery and Development: -

Drug discovery and development is a scientific process through which new medicines are identified, evaluated, and brought to the market for therapeutic use. It is a highly complex and multidisciplinary field involving chemistry, biology, pharmacology, and medical sciences. The aim is to develop drugs that are safe, effective, and of high quality for the treatment and prevention of diseases such as Cancer and infectious disorders.

Drug discovery and development is a systematic and lengthy process used to identify, test, and bring new medicines to market. It begins with the

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identification of a biological target such as a protein, gene, or receptor that is associated with a disease. Once a target is identified, scientists validate it to confirm that modifying it will have a therapeutic effect. After validation, the process moves to the discovery phase where thousands of chemical compounds are screened to find "hit" molecules that show potential activity against the target.

These hit compounds are then optimized to improve their effectiveness, safety, and drug-like properties, leading to the selection of a promising "lead" compound. The next stage is preclinical testing, where the lead compound is tested in laboratory experiments and animal models to evaluate its toxicity, pharmacokinetics, and overall safety profile. If the results are satisfactory, the drug enters clinical trials, which are conducted in humans.

Clinical trials are carried out in three main phases. Phase I trials involve a small number of healthy volunteers to assess safety and dosage. Phase II trials are conducted on a larger group of patients to evaluate the drug's effectiveness and side effects. Phase III trials include a much larger population to confirm effectiveness, monitor adverse reactions, and compare the new drug with existing treatments. If the clinical trials are successful, the data is submitted to regulatory authorities for approval.

Regulatory agencies such as the FDA or CDSCO carefully review all preclinical and clinical data to ensure the drug is safe and effective for public use. Once approved, the drug is manufactured on a large scale and marketed for medical use. Even after approval, the drug continues to be monitored in the post-marketing or Phase IV stage to detect any long-term or rare side effects in the general population.

1.2 Drug Development Stages: -

Figure no.1 Drug Development Stages

Drug development is a long and complex process used to discover, test, and bring a new medicine to market. It ensures that drugs are safe, effective, and of good quality before being used by patients.

1. Drug Discovery & Development

This is the initial stage where scientists identify a disease target (such as a protein or gene) and search for potential drug molecules.

- Target identification (finding disease-related molecules)

- Target validation (confirming its role in disease)
- Screening of thousands of compounds
- Selection of promising "hit" compounds

2. Preclinical Research

Before testing in humans, the drug is tested in the laboratory and on animals.

- Laboratory (in vitro) testing
- Animal (in vivo) testing
- Study of toxicity, safety, and dosage
- Evaluation of pharmacokinetics (absorption, distribution, metabolism, excretion)

3. Clinical Trials (Human Testing)

This stage tests the drug in humans and is divided into 3 phases:

🔴 Phase I

Small group (20–100 healthy volunteers)

Focus: Safety and dosage

🟠 Phase II

Larger group (100–300 patients)

Focus: Effectiveness and side effects

🟡 Phase III

Large population (1,000–3,000+ patients)

Focus: Confirmation of effectiveness, monitoring adverse reactions

4. Regulatory Approval

Government agencies review all data to ensure safety and effectiveness.

Agencies: FDA (USA), CDSCO (India), EMA (Europe)

Review of clinical and preclinical data

Approval is granted if standards are met

5. Manufacturing & Marketing

Once approved, the drug is produced on a large scale and made available to the public.

- Large-scale production
- Quality control
- Distribution to pharmacies and hospitals
- Marketing and awareness

6. Post-Market Surveillance (Phase IV)

Even after approval, the drug is continuously monitored.

- Detect rare or long-term side effects
- Monitor safety in large populations
- Improve usage guidelines if needed

1.3 CADD (COMPUTER AIDED DRUG DESING) :-

Computer-Aided Drug Design (CADD) is a computational approach used in modern pharmaceutical research to discover, design, and optimize new drug molecules with the help of computer software and simulation techniques. It plays an important role in reducing the time, cost, and experimental effort required in traditional drug discovery processes. CADD integrates knowledge from chemistry, biology, pharmacology, and

computer science to predict how small molecules interact with biological targets such as proteins, enzymes, or receptors.

CADD is broadly classified into two main types: structure-based drug design (SBDD) and ligand-based drug design (LBDD). Structure-based drug design is used when the three-dimensional structure of the target protein is known. In this method, researchers study the active site of the protein and design or screen molecules that can fit into the binding pocket effectively. Techniques such as molecular docking and molecular dynamics simulation are commonly used to analysed the interaction between the drug and the target protein. Ligand-based drug design is used when the structure of the target protein is not available but information about known active compounds exists. In this approach, scientists analysed existing molecules that show biological activity and identify common chemical features responsible for their effectiveness. Methods such as pharmacophore modelling and quantitative structure–activity relationship (QSAR) are used to predict the activity of new compounds based on structural similarity.

The CADD process typically begins with target identification, where a disease-related biological molecule is selected. This is followed by target structure preparation, in which the three-dimensional structure of the protein is obtained from experimental databases or computational modelling. After this, large chemical libraries containing millions of compounds are prepared for virtual screening. Virtual screening is a computational technique used to filter and identify the most promising drug-like molecules from large databases.

The selected compounds are then subjected to molecular docking studies to predict their binding orientation and affinity with the target protein. The best-performing compounds are further optimized to improve their potency, selectivity, and safety. In addition, ADMET prediction is performed to evaluate absorption, distribution, metabolism, excretion, and toxicity properties of the drug candidates before experimental testing.

CADD is widely used in modern drug discovery for diseases such as cancer, viral infections, bacterial infections, and neurological disorders. It has significantly contributed to the development of antiviral drugs, including drugs used in HIV and COVID-19 treatment research. Pharmaceutical industries extensively use CADD tools such as AutoDock, Schrödinger Suite, MOE, and Discovery Studio for drug screening and optimization. One of the major advantages of CADD is that it reduces the need for extensive laboratory experiments by predicting the most promising compounds in advance.

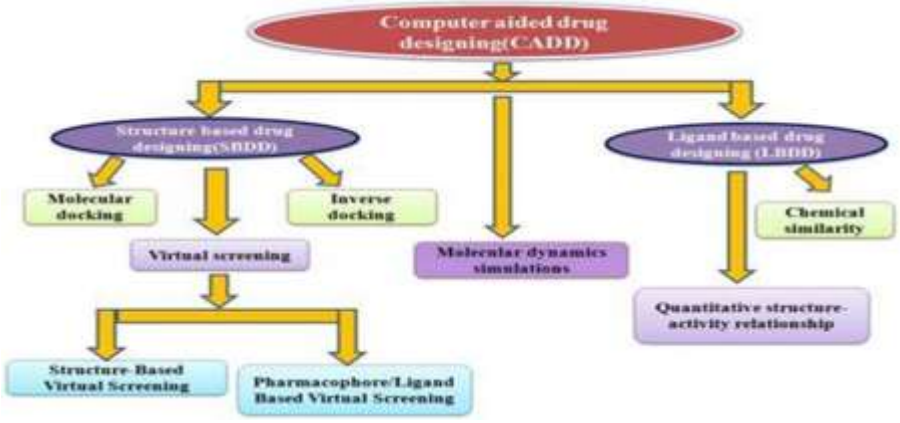


Figure no.2 Computer Aided Drug Desing

1.4 Molecular Docking: -

Molecular docking is a computational technique used in Computer-Aided Drug Design (CADD) to predict the preferred orientation of a small molecule (ligand) when it binds to a target protein or receptor. It helps scientists understand how a drug molecule fits into the active site of a protein and how strong the interaction is. The main goal of molecular docking is to estimate the binding affinity between the ligand and the target, which indicates how effectively a drug can inhibit or activate a biological function.

In molecular docking, both the ligand and the receptor are modelled in three-dimensional form. The ligand is placed into the binding site of the receptor in different orientations, and each position is evaluated using scoring functions. These scoring functions predict the stability and strength of the interaction based on factors such as hydrogen bonding, hydrophobic interactions, electrostatic forces, and van der Waals forces.

Molecular docking is generally divided into two main steps: search algorithm and scoring function. The search algorithm explores possible conformations and orientations of the ligand within the binding site, while the scoring function ranks these conformations based on predicted binding energy. The best docking pose is the one with the lowest binding energy and strongest interaction with the target protein.This technique is widely used in drug discovery to screen large libraries of compounds and identify potential drug candidates quickly and efficiently. It reduces the need for extensive laboratory experiments by narrowing down the most promising molecules for further testing. Molecular docking is especially useful in the development of drugs for cancer, infectious diseases, and neurological disorders.

- Types of Molecular Docking

Molecular docking is classified based on the flexibility of the ligand (drug molecule) and the receptor (target protein). The main types are

1. Rigid Docking

2. Flexible Ligand Docking

3. Flexible Receptor Docking

4. Induced Fit Docking

5. Ensemble Docking

🟦 1. Rigid Docking

In rigid docking, both the ligand and the receptor are treated as completely rigid structures. No flexibility is allowed in either molecule during docking. The ligand is simply fitted into the fixed active site of the receptor. This method is fast and computationally simple but less accurate because it does not reflect real biological conditions.

🟧 2. Flexible Ligand Docking

In flexible docking, the ligand is allowed to rotate and change its shape while binding to the receptor. The receptor remains rigid in most cases. This method provides more realistic results compared to rigid docking because it considers different conformations of the ligand.

🟪 3. Flexible Receptor Docking

In this method, the receptor protein is also allowed some flexibility during docking. This is important because proteins can change shape when a ligand binds to them. However, this method is more complex and requires higher computational power.

🟩 4. Induced Fit Docking

Induced fit docking allows flexibility in both the ligand and the receptor. It considers that the

protein binding site may adjust its shape to fit the ligand. This method is more accurate and closely represents real biological interactions but is computationally expensive.

🟧 5. Ensemble Docking

In ensemble docking, multiple different conformations of the receptor are used for docking. This helps in considering protein flexibility without changing its structure during simulation. It improves prediction accuracy and is widely used in modern drug discovery.

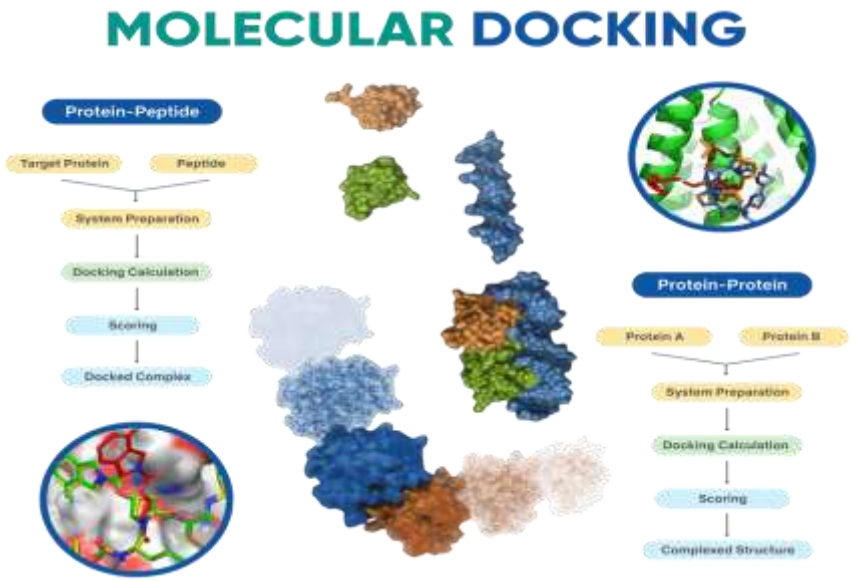


Figure no.3 Molecular Docking

1.5 Wound Healing: -

Wound healing is a natural biological process by which the body repairs damaged tissues after injury. It involves a complex series of events that restore the integrity and function of the skin or other tissues. The process is essential for survival because it prevents infection, reduces blood loss, and restores normal tissue structure. Wound healing occurs in four main phases: hemostasia, inflammation, proliferation, and remodelling.

The first stage, hemostasia, begins immediately after injury, where blood vessels constrict and blood clotting occurs to stop bleeding. Platelets play an important role by forming a fibrin clot that acts as a temporary barrier.

The second stage is the inflammatory phase, where white blood cells such as neutrophils and macrophages migrate to the wound site to remove dead cells, bacteria, and debris. This phase helps prevent infection and prepares the wound for healing.

The third stage is the proliferative phase, where new tissue is formed. Fibroblasts produce collagen, which provides strength to the wound. New blood vessels form through angiogenesis, and the wound begins to close as epithelial cells grow over the damaged area.

The final stage is the remodelling or maturation phase, which can last for months or even years. During this phase, collagen fibres are reorganized and strengthened, and the wound tissue gradually gains maximum strength, although it may never fully regain the original strength of the skin.

Wound healing can be affected by factors such as age, nutrition, blood supply, infection, diabetes, and medications. Poor healing may lead to chronic

wounds or complications such as ulcers. Wound healing is an important area of medical research, and various drugs, dressings, and biomaterials are developed to enhance speed up the healing process.

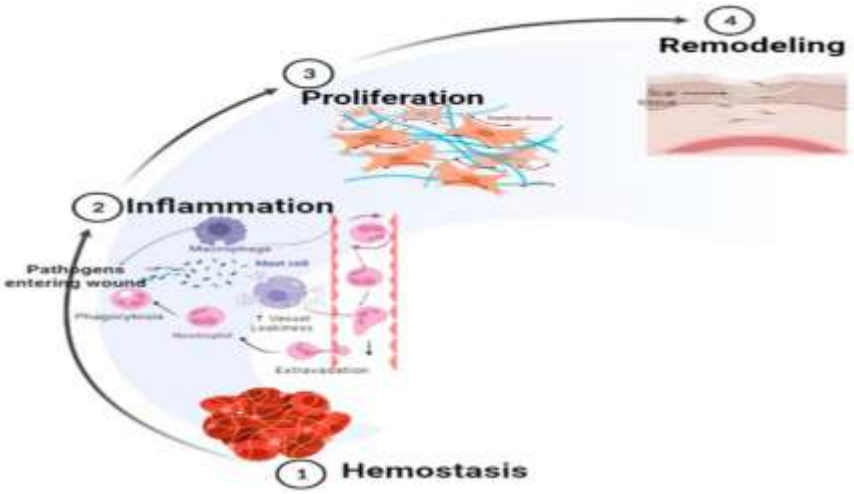


Figure no.4 Mechanism of Action of Wound Healing

Receptors Involved in Wound Healing

Wound healing is regulated by several cell surface receptors that respond to growth factors, cytokines, and extracellular signals. These receptors control inflammation, cell migration, tissue formation, and remodelling during the healing process.

- One of the most important receptors involved is the Interleukin-6 receptor (IL-6R). IL-6R binds interleukin-6 and activates the JAK/STAT signalling pathway, which regulates inflammation, immune cell activation, and tissue repair. This receptor plays a key role in the early inflammatory phase and also supports tissue regeneration.

- Another important receptor is the Toll-like receptors (TLRs), which are found on immune cells such as macrophages and dendritic cells. TLRs recognize pathogens and damaged tissue signals, triggering the release of cytokines that initiate the inflammatory response required for wound healing.

- The Transforming Growth Factor-beta (TGF-β) receptor is also crucial in wound repair. It regulates fibroblast activation, collagen synthesis, and extracellular matrix production. TGF-β signalling is essential for tissue formation and remodelling phases of healing.

- The Vascular Endothelial Growth Factor (VEGF) receptor plays a major role in angiogenesis, which is the formation of new blood vessels. VEGF receptor activation ensures oxygen and nutrient supply to the healing tissue, promoting faster recovery.

- The Platelet-Derived Growth Factor (PDGF) receptor stimulates the migration and proliferation of fibroblasts and smooth muscle cells. It contributes to tissue regeneration and wound closure.

- In addition, Epidermal Growth Factor (EGF) receptors are involved in the proliferation and migration of keratinocytes, which helps in re-epithelialization of the wound surface.

- Overall, these receptors work together in a coordinated manner to control inflammation, cell growth, blood vessel formation, and tissue remodelling, ensuring proper wound healing.

Role of IL-6 Receptor in Wound Healing

Interleukin-6 (IL-6) is a multifunctional cytokine that plays an important role in regulating inflammation and tissue repair during wound healing. The IL-6 receptor (IL-6R) is a cell surface receptor that binds IL-6 and activates intracellular signalling pathways involved in immune response and tissue regeneration. IL-6 signalling occurs through two main mechanisms: classic signalling and trans-signalling.

In classic signalling, IL-6 binds to membrane-bound IL-6R present on certain cells such as hepatocytes and some immune cells. This complex then associates with a signal-transducing protein called gp130, leading to activation of intracellular pathways such as JAK/STAT3, MAPK, and PI3K/AKT. These pathways regulate cell survival, proliferation, and inflammation, which are essential for wound repair. In trans-signalling, IL-6 binds to a soluble form of IL-6R, and this complex can act on a wider range of cells that do not normally express IL-6R. This mechanism is particularly important in chronic inflammation and tissue regeneration during wound healing.

During the early inflammatory phase of wound healing, IL-6 is rapidly produced by immune cells such as macrophages, neutrophils, and fibroblasts. It helps recruit additional immune cells to the wound site and promotes the clearance of pathogens and damaged tissue. IL-6 also stimulates acute phase responses that support the body's Défense mechanisms.

In the proliferative phase, IL-6 contributes to tissue regeneration by promoting fibroblast activation, collagen production, and angiogenesis (formation of new blood vessels). It also supports keratinocyte migration, which is important for re-epithelialization of the wound surface. However, excessive or prolonged IL-6 signalling can lead to chronic inflammation and delayed wound healing. Elevated IL-6 levels are often associated with non-healing wounds such as diabetic ulcers and inflammatory skin conditions. Overall, the IL-6 receptor plays a dual role in wound healing by promoting both inflammatory Défense and tissue repair. Proper regulation of IL-6 signalling is essential for balanced and effective wound healing.

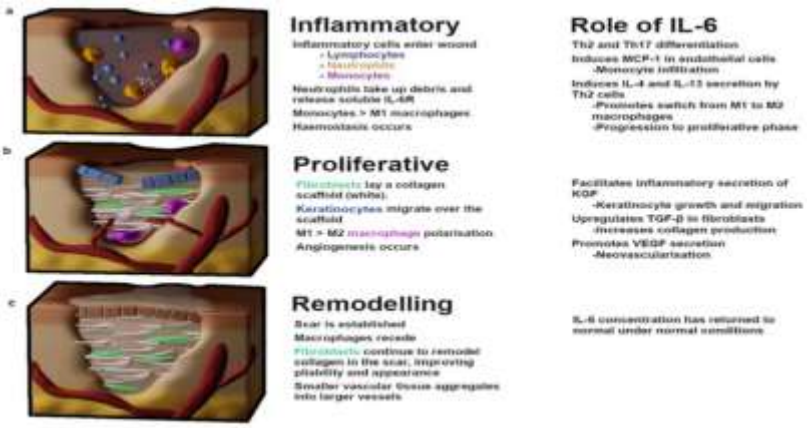


Figure no.5 IL-6 Mechanism of Wound Healing

1.6 Tridax Procumbens: -

📌 Biological Source

Tridax procumbens consists of the whole plant or leaves of Tridax procumbens Linn., belonging to the family Asteraceae.

🌍 Geographical Source

The plant is widely distributed in tropical and subtropical regions, especially in India, Africa, and South America. It commonly grows as a weed in fields, roadsides, and waste lands.

🌱 Macroscopic Characters

- Leaves: Opposite, simple, hairy, irregularly toothed margins
- Stem: Weak, branched, hairy, creeping or ascending
- Flowers: Yellow disc florets with white ray florets (daisy-like appearance)
- Roots: Taproot system
- Odor: Characteristic
- Taste: Slightly bitter

🔬 Microscopic Characters

- Presence of multicellular covering trichomes
- Epidermal cells with cuticle
- Vascular bundles (xylem and phloem) well developed
- Calcium oxalate crystals may be present
- Parenchyma cells containing starch grains

🧪 Chemical Constituents

- Flavonoids (quercetin, luteolin)
- Alkaloids
- Tannins
- Saponins
- Carotenoids
- Steroids (β-sitosterol)

💊 Pharmacological Actions

- Wound healing activity
- Anti-inflammatory
- Antimicrobial
- Antioxidant
- Hepatoprotective
- Antidiabetic

⚙️ Mechanism of Action (Wound Healing)

The plant promotes wound healing by:

- Increasing collagen synthesis
- Enhancing fibroblast proliferation
- Promoting angiogenesis
- Accelerating epithelialization
- Reducing inflammation and microbial infection

💊 Uses

- Treatment of wounds and cuts
- Stops bleeding (hemostatic property)
- Skin infections

- Anti-inflammatory applications

Preparation / Formulation

- Leaf paste applied externally
- Extracts used in ointments and gels
- Herbal formulations for wound healing

Figure no.6 Tridax Procumbens Plant

2. AIM AND OBJECTIVE: -

AIM: -

To investigate the pharmacological potential of Tridax procumbens in wound healing by studying its effects on collagen synthesis, fibroblast proliferation, angiogenesis, and epithelialization.

OBJECTIVES : -

- To study the wound healing properties of Tridax procumbens.
- To evaluate the effect of Tridax procumbens on collagen synthesis and tissue regeneration.
- To analysed the anti-inflammatory activity of Tridax procumbens in wound healing.
- To investigate the role of Tridax procumbens in promoting fibroblast proliferation and angiogenesis.
- To assess the antimicrobial activity of Tridax procumbens in preventing wound infection.
- To determine the overall effectiveness of Tridax procumbens in accelerating the wound healing process.

3. EXPERIMENT WORK: -

3.1 Downloading Software Program: -

PYRX: - PyRx is an open-source virtual screening software used in Computer-Aided Drug Design to perform molecular docking and analysed ligand–protein interactions. It provides a user-friendly graphical interface that integrates tools such as AutoDock and AutoDock Vina for docking simulations. PyRx allows researchers to import protein and ligand structures, prepare molecules, and perform energy minimization before docking.

The software is widely used for virtual screening of large compound libraries to identify potential drug candidates. It helps in predicting binding affinity and selecting the best ligand based on docking scores. PyRx also supports visualization of molecular interactions, which aids in understanding how a drug binds to its target receptor.

BIOVIA-DISCOVERY STUDIO: - BIOVIA Discovery Studio is widely used in Computer-Aided Drug Design (CADD) to study interactions between ligands (drug molecules) and biological targets such as proteins and enzymes. It allows researchers to visualize molecular structures in three dimensions and analysed binding interactions at the atomic level.

The software includes various modules for molecular docking, pharmacophore modelling, quantitative structure–activity relationship (QSAR) studies, and virtual screening.

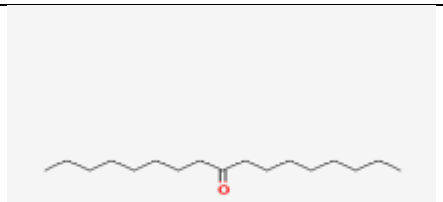
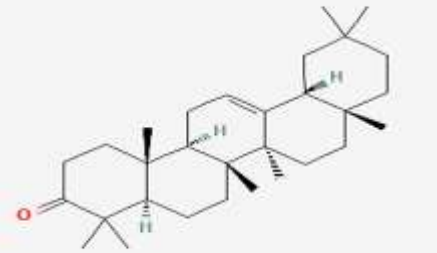
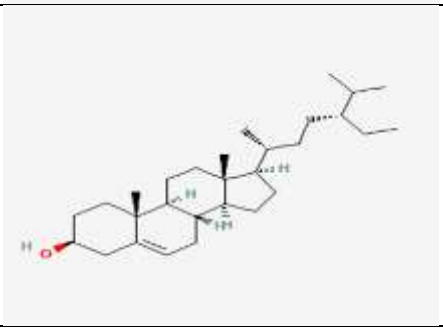
AVAGADRO**: -** is an open-source molecular modelling and visualization tool used in chemistry, pharmacology, and Computer-Aided Drug Design (CADD). It is designed to build, edit, and visualize molecular structures in three-dimensional (3D) form.

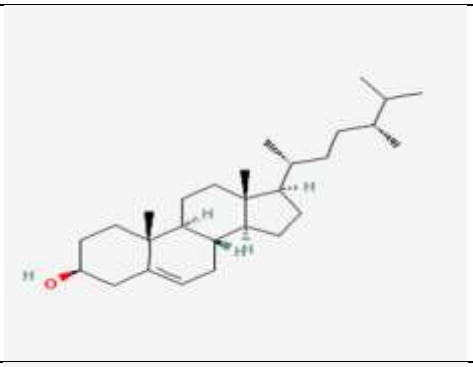
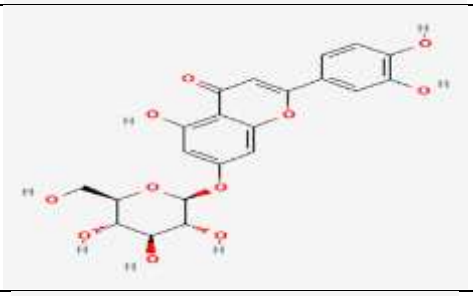
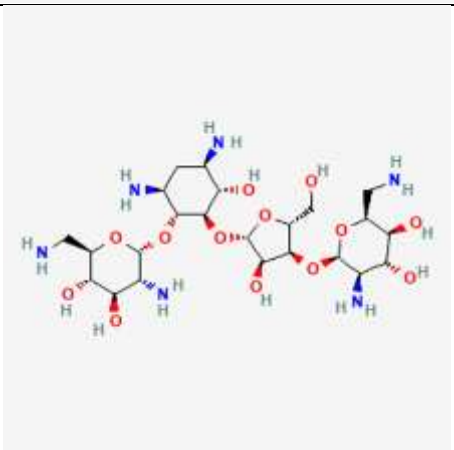
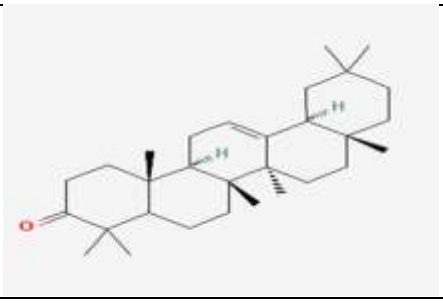
CHEMSKETCH: **-** ACD/ChemSketch is a chemical drawing and molecular modeling software developed by Advanced Chemistry Development (ACD/Labs). It is widely used in chemistry, pharmacology, and Computer-Aided Drug Design (CADD) for drawing chemical structures and predicting basic molecular properties.

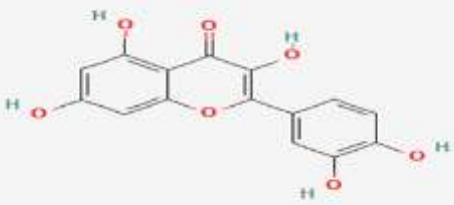
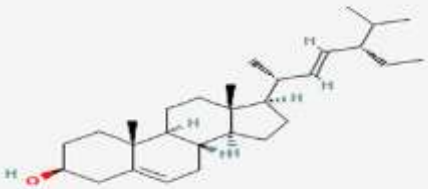
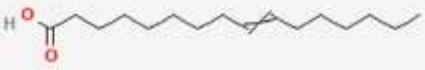
3.2 Preparation of Ligand: -

In this study, Triadx Procumbens, was chosen for wound healing properties. Various phytochemical present in triadx procumbens to its therapeutic effects. To understand further select phytoconstituent using Chemsketch software and then structure was cleaned and then structure was saved in the working folder as mol file. This mol file was then accessed in Avogadro software tool in which that the mol file is convert to pdb format and then structure was optimized by using the optimization tool and then saved the optimized structure in the working directory as .pdb file.

Table No.1. Phytoconstituent of Tridax Procumbens

Sr.No	Ligands	IUPAC Name	2D Structure
1	9-Heptadecanone	heptadecan-9-one	
2	Beta-amyrone	4aR,6aR,6bS,8aR,12aR,14aR,14bR)-4,4,6a,6b,8a,11,11,14b-octamethyl-2,4a,5,6,7,8,9,10,12,12a,14,14a-dodecahydro-1H-picen-3-one	
3	Beta-sitosterol	(3S,8S,9S,10R,13R,14S,17R)-17-[(2R,5R)-5-ethyl-6-methylheptan-2-yl]-10,13-dimethyl-2,3,4,7,8,9,11,12,14,15,16,17-dodecahydro-1H-cyclopenta[a]phenanthren-3-ol	

4	Campesterol	: (3S,8S,9S,10R,13R,14S,17R)-17-[(2R,5R)-5,6-dimethylheptan-2-yl]-10,13-dimethyl-2,3,4,7,8,9,11,12,14,15,16,17-dodecahydro-1H-cyclopenta[a] phenanthren-3-ol	
5	Cynaroside	2-(3,4-dihydroxyphenyl)-5-hydroxy-7-[(2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl) oxan-2-yl] oxychromen-4-one	
6	Framycetin	(2R,3S,4R,5R,6R)-5-amino-2-(aminomethyl) -6-[(1R,2R,3S,4R,6S)-4,6-diamino-2-[(2S,3R,4S,5R)-4-[(2R,3R,4R,5S,6S) -3-amino-6-(aminomethyl) -4,5-dihydroxyoxan-2-yl] oxy-3-hydroxy-5-(hydroxymethyl)oxolan-2-yl] oxy-3-hydroxycyclohexyl] oxyoxane-3,4-diol	
7	Lupeol	(1R,3aR,5aR,5bR,7aR,9S,11aR,11bR, 13aR,13bR)-3a,5a,5b,8,8,11a-hexamethyl-1-prop-1-en-2-yl-1,2,3,4,5,6,7,7a,9,10,11,11b,12,13, 13a,13b-hexadecahydrocyclopenta [a]chrysen-9-ol	
8	Olane-12-en-3-one	(6aR,6bS,8aR,12aS,14aR,14bR) -4,4,6a,6b,8a,11,11,14b-octamethyl-2,4a,5,6,7,8,9,10,12,12a,14,14a-dodecahydro-1H-picen-3-one	

9	Quercetin	2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxychromen-4-one	
10	Stigmasterol	(3S,8S,9S,10R,13R,14S,17R)-17-[(E,2R,5S)-5-ethyl-6-methylhept-3-en-2-yl]-10,13-dimethyl-2,3,4,7,8,9,11,12,14,15,16,17-dodecahydro-1H-cyclopenta[a]phenanthren-3-ol	
11	Linoleic	octadeca-9,12-dienoic acid	
12	Linolenic	octadeca-9,12,15-trienoic acid	
13	Palmitoleic acid	hexadec-9-enoic acid	

3.3 Preparation of Receptor: -

- The download PDB structure (6P9E) was opened in Discovery Studio Visualizer.
- All water molecule, heteroatoms, and co-crystallized ligand were removed to clean the receptor.
- Polar hydrogen atom were added to stabilized the protein structure.
- The active site residues were defined based on the known Wound healing binding pocket.
- The final receptor file was saved in PDBQT format using AutoDock tools for compatibility with docking software.

Figure no.7 3ED Structure of receptor 6P9E

3.4 Physicochemical Properties: -

Lipinski rule was used to assess the physiochemical properties of all the selected ligands and to predict their drug like properties, and the Swiss ADME as used to computer SMILE structure of each compound.

3.5 ADME Studies:

ADME (Absorption, Distribution, Metabolism and Excretion) studies are indeed crucial in drug development to assess how a drug behaves the body. Swiss ADME software was used to determine these properties of each ligand.

3.6 Druglikeness :-

SwissADME evaluates drug-likeness by applying rules such as Lipinski, Ghose, Veber, Egan, and Muegge, along with physicochemical properties like molecular weight, lipophilicity, and polarity, to predict whether a compound is suitable for oral drug development.

3.7 Toxicity Study: -

It allows users to input a compound (via name, SMILES, or structure) and predicts over 60 toxicity endpoints, including acute toxicity (LD_{50}), organ toxicity (like hepatotoxicity and neurotoxicity), carcinogenicity, mutagenicity, immunotoxicity, and clinical toxicity.

The platform uses multiple predictive models (about 61) based on approaches such as fragment analysis, pharmacophore modelling, and machine learning algorithms (e.g., Random Forest and neural networks) to generate results with confidence scores.

3.8 Molecular Docking: -

To perform molecular docking using PyRx:-

- Load protein and ligand files
- Perform ligand energy minimization
- Convert protein to macromolecule (PDBQT)
- Convert ligand to ligand format (PDBQT)
- Open Vina Wizard
- Select macromolecule and ligand
- Set grid box parameters

- Run docking using AutoDock Vina
- Analysed binding energy and poses
- Save/export docking results

4. RESULTS AND DISCUSSION: -

1. Physicochemical properties: -

The physicochemical properties of the compound were studied to predict the pharmacokinetics of the drugs, using Lipinski's Rule of Five is a set of guidelines used to evaluate the drug-likeness of a compound, particularly its potential for good oral bioavailability. It states that a molecule is more likely to be orally active if it has a molecular weight of 500 Daltons or less, a lipophilicity value (Log P) not greater than 5, no more than 5 hydrogen bond donors, and no more than 10 hydrogen bond acceptors.

Table No.2. Physicochemical Properties of Ligands

Sr. No	Ligands	No. of rotatable bonds	No. of H-bond accept	No. of H-bond donors	Molar refractivity	Molecular Weight (g/mol)	TPSA
1	9-Heptadecanone	14	1	0	84.03	254.45	17.07
2	Beta-amyrin	0	1	1	134.88	426.72	20.23
3	Beta-sitosterol	6	1	1	133.23	414.71	20.23
4	Campesterol	5	1	1	128.42	400.68	20.23
5	Cynarocide	4	11	7	108.13	448.38	190.28
6	Framycetin	9	19	13	135.11	614.64	353.11
7	Lupeol	1	1	1	135.14	426.72	20.23
8	Olane-12-en-3-one	0	1	10	132.92	424.70	17.07
9	Quercetin	1	7	5	78.03	302.24	131.36
10	Stigmasterol	5	1	1	132.75	412.69	20.23
11	Linoleic	14	2	1	89.46	280.85	37.30
12	Linolenic	13	2	1	88.99	278.43	37.30
13	Palmitoleic acid	13	2	1	80.32	254.41	37.30

2. ADME Properties: -

ADME data predict using the Swiss ADME online web server database of phytocompounds.

Table No.3. Pharmacokinetics Properties of Ligands

Sr. No	Ligands	GI absorption	BBB permeant	P-gp substrate	CYP1A2 inhibitor	CYP2C19 inhibitor	Log Kp (cm/s)
1	9-Heptadecanone	HIGH	NO	NO	YES	NO	-2.87
2	Beta-amyrin	LOW	NO	NO	NO	NO	-2.41
3	Beta-sitosterol	LOW	NO	NO	NO	NO	-2.20
4	Campesterol	LOW	NO	NO	NO	NO	-2.50
5	Cynarocide	LOW	NO	YES	NO	NO	-8.00
6	Framycetin	LOW	NO	YES	NO	NO	-16.43
7	Lupeol	LOW	NO	NO	NO	NO	-1.90
8	Olane-12-en-3-one	LOW	NO	NO	NO	NO	-2.61
9	Quercetin	HIGH	NO	NO	YES	NO	-7.05
10	Stigmasterol	LOW	NO	NO	NO	NO	-2.74
11	Linoleic	HIGH	YES	NO	YES	NO	-3.05
12	Linolenic	HIGH	YES	NO	YES	NO	-3.41

13	Palmitoleic acid	HIGH	YES	NO	YES	NO	-3.18

3. Drug likeness :-

The drug-likeness capability of phytoconstituents can be predict using Lipinski, Ghose, Veber, Egan, and Muegge rules which are based on certain physicochemical parameters.

Table No.4. Drug likeness Properties of Ligands

Sr. No	Ligands	Lipinski	Ghose	Veber	Egan	Muegge	Bioavailability score
1	9-Heptadecanone	YES	NO	NO	NO	NO	0.55
2	Beta-amyrone	YES	NO	YES	NO	NO	0.55
3	Beta-sitosterol	YES	NO	YES	NO	NO	0.55
4	Campesterol	YES	NO	YES	NO	NO	0.55
5	Cynarocide	NO	YES	NO	NO	NO	0.17
6	Framycetin	NO	NO	NO	NO	NO	0.17
7	Lupeol	YES	NO	YES	NO	NO	0.55
8	Olane-12-en-3-one	YES	NO	YES	NO	NO	0.55
9	Quercetin	YES	YES	YES	YES	YES	0.55
10	Stigmasterol	YES	NO	YES	NO	NO	0.55
11	Linoleic	YES	NO	NO	NO	NO	0.85
12	Linolenic	YES	NO	NO	YES	NO	0.85
13	Palmitoleic acid	YES	YES	NO	YES	NO	0.85

4. Toxicity Study :-

Toxicity prediction is the important step in drug development and design. High demand for computational predictive model to evaluate the potent toxic effects of drugs. In silico study toxicity of drug is evaluating by Protox 3.0 online database.

Table No.5. Toxicity Study of Ligand

Sr. No	Ligands	Predicted Toxicity class	Predicted LD50 (mg/kg)	Carcinogenicity	Hepato toxicity	Immuno toxicity	Nephro toxicity
1	9-Heptadecanone	5	5000	INACTIVE	INACTIVE	INACTIVE	INACTIVE
2	Beta-amyrone	5	5000	INACTIVE	INACTIVE	ACTIVE	INACTIVE
3	Beta-sitosterol	4	890	INACTIVE	INACTIVE	ACTIVE	INACTIVE
4	Campesterol	4	890	INACTIVE	INACTIVE	ACTIVE	INACTIVE
5	Cynaroside	5	5000	INACTIVE	INACTIVE	INACTIVE	ACTIVE
6	Framycetin	5	2275	INACTIVE	INACTIVE	INACTIVE	ACTIVE
7	Lupeol	4	2000	INACTIVE	INACTIVE	ACTIVE	INACTIVE
8	Olane-12-en-3-one	5	5000	INACTIVE	INACTIVE	ACTIVE	INACTIVE
9	Quercetin	3	159	ACTIVE	INACTIVE	INACTIVE	INACTIVE
10	Stigmasterol	4	890	INACTIVE	INACTIVE	ACTIVE	INACTIVE
11	Linoleic	4	1190	INACTIVE	ACTIVE	ACTIVE	INACTIVE
12	Linolenic	6	10000	INACTIVE	INACTIVE	INACTIVE	INACTIVE
13	Palmitoleic acid	2	48	INACTIVE	INACTIVE	INACTIVE	INACTIVE

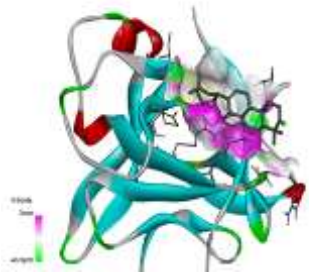
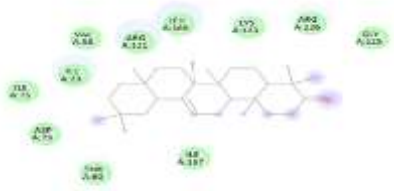
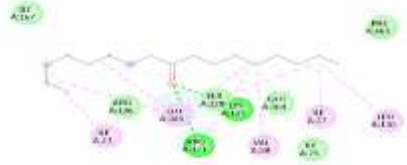
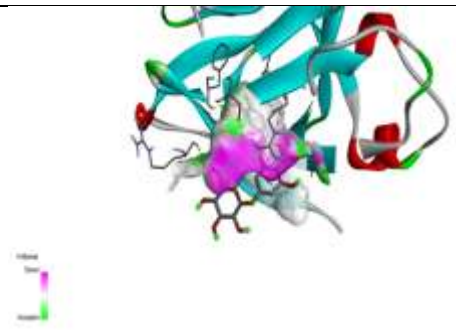
5. Binding affinity of Ligands :-

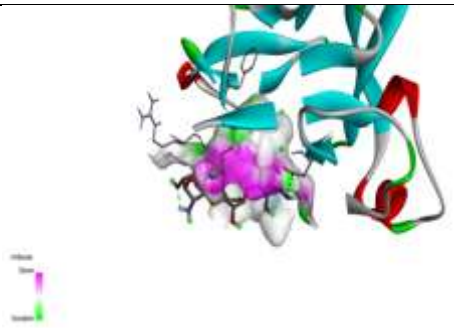
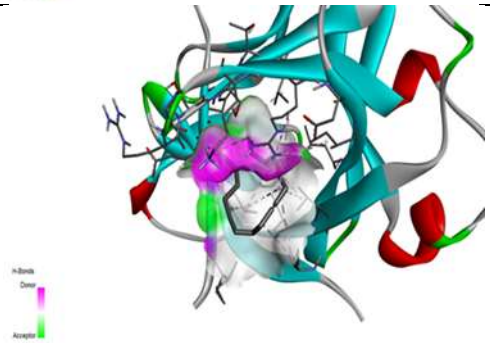
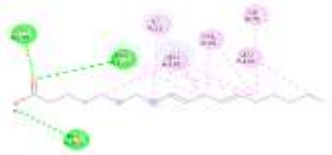
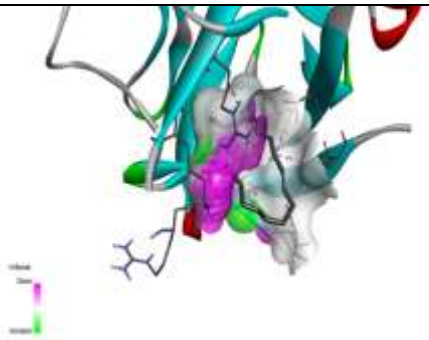
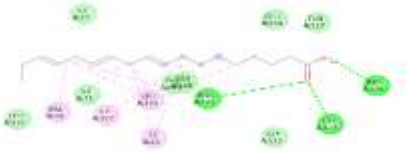
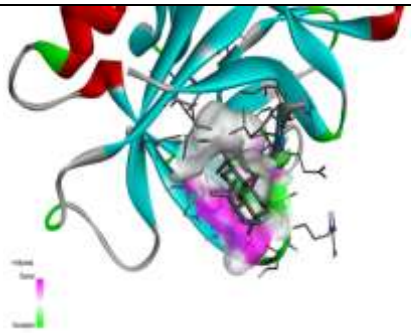
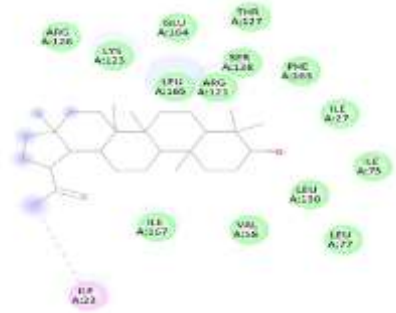
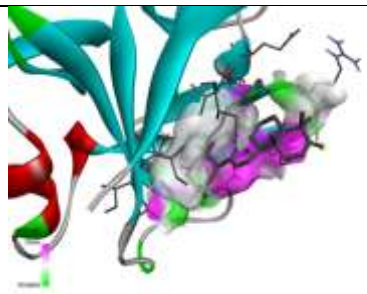
Table No.6. Binding affinity of phytoconstituents of Triadx Procumbens with (IL-6) 9P6E receptor

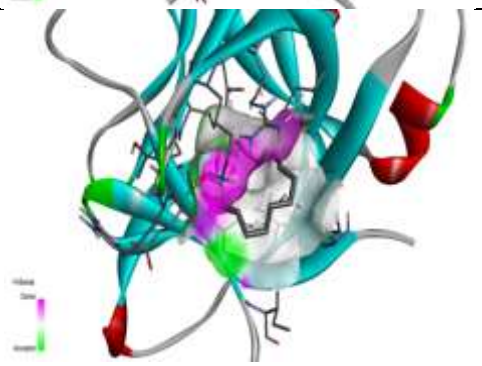
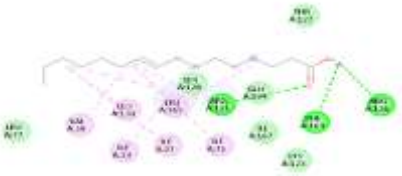
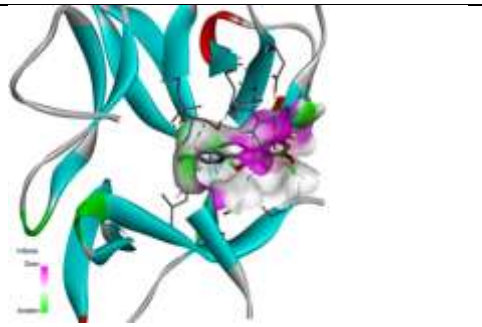
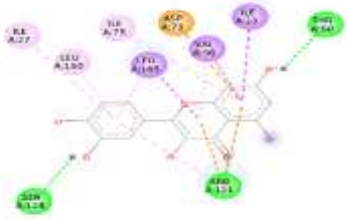
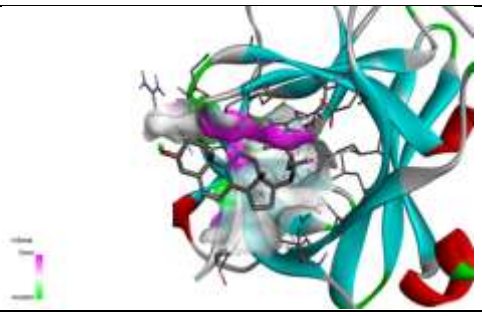
Sr. No	Ligands	Binding affinity
1	9-Heptadecanone	-5.4
2	Beta-amyrone	0.5
3	Beta-sitosterol	-
4	Campesterol	-5.9
5	Cynaroside	-7.7
6	Framycetin	-5.3
7	Lupeol	-3.6
8	Olane-12-en-3-one	-1.9
9	Quercetin	-7.3
10	Stigmasterol	-5.1
11	Linoleic	-6.1
12	Linolenic	-6.2
13	Palmitoleic acid	-6.1

6. 3D and 2D Structure of Phytoconstituents of Tridax Procumbens: -

Table no.7. 3D and 2D Structures of Phytoconstituents of Tridax Procumbens

Sr.No	LIGAND	3D STRUCTURE	2D STRUCTURE
1.	BETA-AMYRONE		
2.	9-HEPTADECACONE		
3.	CAMPESTEROL		
4.	CYNAROSIDE		

5.	FRAMY CETINE		
6.	LINOLEIC		
7.	LINOLENIC		
8.	LUPIOL		
9.	OLANE-12-EN-3-ONE		

10.	PALMITIC		
11.	PALMITOLIC ACID		
12.	QUERCITIN		
13.	STIGME STEROL		

5. CONCLUSION: -

The Tridax Procumbens contain 13 majors Phytoconstituents. The study of ADME showed all phytoconstituents are absorbed from the GI tract and Toxicity showed they are non-toxic and obey the Lipinski rule. The Molecular Docking of phytoconstituents with 6P9E receptor for Wound Healing showed Cynaroside, Quercetin, Linoleic, Linolenic, Palmitoleic acid, Campesterol, 9-Heptadecanone have good binding affinity as compare as to standard drug

Framycetin, therefore Cynaroside, Quercetin, Linoleic, Linolenic, Palmitoleic acid, Campesterol, 9-Heptadecanone act as lead molecule for further development on acting IL-6 receptor for better Wound Healing activity with minimum sides effects.

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