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MOLECULAR DOCKING ANALYSIS OF THE BIOACTIVE COMPOUND EMODIN WITH VEGFR2 IN CERVICAL CANCER

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ABSTRAK

Kanker serviks merupakan salah satu penyebab utama kematian akibat kanker pada perempuan dan menjadi kanker terbanyak ketiga di dunia. Ekspresi VEGF yang tinggi pada kanker serviks dikaitkan dengan prognosis yang buruk, sehingga terapi anti-VEGF mempunyai peran penting dalam penyembuhan kanker serviks. Hasil beberapa penelitian menunjukkan emodin yang merupakan turunan antrakuinon alami terutama diisolasi dari Rheum palmatum menghambat proliferasi, migrasi, dan pembentukan sel endotel yang distimulasi oleh VEGF. Penelitian ini bertujuan untuk mengetahui interaksi dari senyawa bioaktif emodin terhadap reseptor VEGF pada kanker serviks secara in silico dengan molecular docking. Molecular docking dilakukan dengan beberapa tahapan mulai dari preparasi struktur senyawa bioaktif, preparasi struktur protein target, validasi metode molecular docking, dan docking senyawa bioaktif pada protein target. Nilai energi ikatan yang semakin rendah antara senyawa bioaktif dan protein target menunjukkan ikatan yang terbentuk semakin kuat dan stabil. Hasil docking menunjukkan nilai pengikatan dari kompleks VEGFR2-emodin sebesar -7,78 kkal/mol dengan tiga ikatan hydrogen dengan asam amino GLU915, CYS917, LEU838 dan tiga van der Waals dengan asam amino PHE916, GLY920, VAL 846. Terdapat interaksi antara senyawa bioaktif emodin dengan VEGFR2 ditandai dengan nilai pengikatan yang baik (-7,78 kkal/mol) serta membentuk ikatan yang stabil ditandai dengan 3 ikatan hidorgen dan 3 interaksi van der waals.

ABSTRACT

Molecular Docking Analysis of The Bioactive Compound Emodin with VEGFR2 in Cervical Cancer. Cervical cancer remains a leading cause of cancer-related mortality among women and represents the third most common cancer globally. Elevated VEGF expression in cervical cancer is associated with poor prognosis, highlighting the relevance of anti-VEGF therapeutic strategies. Several studies have shown that emodin, a natural anthraquinone derivative mainly isolated from Rheum palmatum, inhibits VEGF-stimulated proliferation, migration, and formation of endothelial cells. This study aims to characterize the interaction between emodin and the VEGF receptor in cervical cancer through in silico molecular docking. The workflow included preparation of the compound structure, preparation of the target protein structure, docking protocol validation, and docking of the compound to the receptor. Lower binding energy reflects a stronger and more stable interaction. The docking analysis showed that the emodin–VEGFR2 complex exhibits a binding energy of −7.78 kcal/mol, forming three hydrogen bonds with GLU915, CYS917, and LEU838, as well as three van der Waals interactions with PHE916, GLY920, and VAL846. These findings demonstrate a stable interaction between emodin and VEGFR2 and suggest its potential as a lead compound for further development of VEGFR2-targeted inhibitors, underscoring the need for subsequent in vitro and in vivo validation.

Introduction

Cervical cancer is one of the leading causes of cancer deaths in women worldwide, with more than 660,000 new cases and 350,000 deaths in 2022.[1] The disease is mostly caused by HPV types 16 and 18, which account for 70% of all cervical cancer cases.[2] In Indonesia, cervical cancer ranks second as the most common cancer, with 36,964 new cases in 2022.[3] Currently available treatments for cervical cancer include radiotherapy, chemotherapy, and immunotherapy.[4] One of the drugs used in cervical cancer therapy is bevacizumab, an anti-angiogenic agent that inhibits tumor neovascularization.[5] However, resistance to oncological drugs, including bevacizumab, frequently emerges and reduces treatment efficacy.[6]

VEGF (Vascular Endothelial Growth Factor) plays an important role in tumor angiogenesis and is a prognostic biomarker in cervical cancer. Anti-VEGF therapy can inhibit neovascularization, thereby inhibiting tumor growth.[7] Emodin itself is a natural compound found in plants and fungi, such as Rheum palmatum and Aspergillus.[8] Emodin shows potential as an anti-VEGF with anti-angiogenic activity and cytotoxicity against cancer cells, including cervical cancer.[9]

The therapeutic limitations associated with monoclonal antibodies underscore the need for alternative strategies. Small-molecule inhibitors such as emodin may offer mechanistic advantages, including the ability to target intracellular regions of VEGFR2 that antibodies cannot access, potential to circumvent resistance mechanisms linked to altered VEGF availability or antibody binding, and generally lower production complexity.

Computational methods such as molecular docking can be used to predict the interaction of emodin with VEGF receptors in cervical cancer, which can accelerate the discovery of alternative therapies.[10] This approach is expected to provide a solution to overcome resistance to existing cancer therapies. Due to the lack of research on the interaction of emodin with VEGF receptors, this study was conducted.

Methods

This study is an experimental study with a computational approach. The study was conducted from July to November 2024 using a laptop with the following specifications: Intel Core™ i5-10300H Processor 2.5 GHZ (8M Cache, up to 4.5 GHz, 4 cores), 8 GB RAM, and Windows 10 operating system.

The servers used to predict target receptors and ligands were SwissADME (www.swissadme.ch/index.php) and SCFBio (www.scfbioiitd.res.in/software/drugdesign/lipinski.jsp#anchortag). The software used for protein and ligand preparation, docking, analysis and visualization of docking results included MarvinView, AutoDockTools version 4.2.6, and Discovery Studio 2016. The research objects consisted of secondary receptor-ligand data obtained from the PDB website (https://www.rcsb.org/) and ligand chemical structure data obtained from the PubChem website (https://pubchem.ncbi.nlm.nih.gov/).

The protein structure used was VEGFR2 (PDB ID: 1Y6A), and the chemical structure of the ligand used was emodin (PubChem CID: 3220).

The independent variables in this study included VEGF receptors, which act as targets for cervical cancer therapy, and emodin ligands, which have the potential to be used for cervical cancer therapy. The dependent variables included the gridbox size used in the simulation and the binding energy value, which describes the strength of the bond between the receptor and the ligand.

Molecular docking was performed using AutoDockTools 1.5.7 and AutoDock 4.2.6. Receptor preparation involved removing water molecules, adding polar hydrogens, and assigning Kollman charges. Ligand preparation included energy minimization, definition of rotatable bonds, and assignment of Gasteiger charges. Method validation was carried out by redocking the native ligand into the VEGFR2 binding site.

Docking simulations were executed using the Lamarckian Genetic Algorithm with 100 runs, a population size of 150, a maximum of 2.5×10^6 energy evaluations, and 27,000 generations, with a mutation rate of 0.02 and a crossover rate of 0.8. The grid box was set to 28 × 38 × 38 Å with a spacing of 1.0 Å, and its center was aligned to the coordinates of the native ligand.

Results

Compound Druglikeness Prediction Results

Druglikeness prediction of compound ligands was performed using the SwissADME and SCFBio websites. On the SwissADME website, the water solubility (LogS) value for emodin was found to be -3.67, indicating that emodin has low solubility and permeability.[11] This is supported by the results of Lipinski's Rule of Five (RO5) calculations performed on the SCFBio website **(Table 1**).

Table 1. Druglikeness analysis results using Lipinski's Rule of Five for potential compounds

Compound	Druglikeness Parameters (Lipinski's rule o five)					Number of Violations
	MW	HBD	HBA	LogP	Mr	
Emodin	270	3	5	1,11	62,67	0

Note : MW (Molecular Weight), HBD (Hydrogen Bond Donor), HBA (Hydrogen Bond Acceptor), LogP (lipophilicity), Mr (Molar Refractivity)

A broader ADMET evaluation, using pkCSM website, provided additional pharmacokinetic insights (**Table 2**). ADMET analysis shows moderate solubility (LogS −3.19) and acceptable intestinal absorption (74.49%), although low Caco-2 permeability and P-gp substrate status suggest efflux may limit bioavailability. Distribution parameters indicate modest tissue penetration and low CNS permeability, reducing the likelihood of central adverse effects. Emodin is not predicted to be a substrate for major CYP isoforms and inhibits only CYP1A2, indicating low risk of metabolic drug–drug interactions.

Clearance is moderate, and renal transporter involvement is minimal. Toxicity predictions are favorable, showing absence of mutagenicity, hepatotoxicity, skin sensitization, and hERG channel inhibition, with an LD50 consistent with moderate acute toxicity.

Table 2. ADMET Summary

Category	Key Parameters	Value
Absorption	Solubility	−3.19
	HIA (%)	74.49
	Caco-2	0.055
	P-gp substrate	Yes
Distribution	VDss (log L/kg)	0.456
	BBB (log BB)	−0.727
Metabolism	CYP substrates	No
	CYP inhibition	Only CYP1A2: Yes
Excretion	Clearance	0.344
Toxicity	AMES	No
	hERG I/II	No
	Hepatotoxicity	No
	LD50 (mol/kg)	2.116

These combined structural and pharmacokinetic characteristics strengthen the plausibility of emodin as a VEGFR2-targeting compound with multi-pathway anticancer potential.

Target Receptor Preparation Results

The VEGFR2 structure (PDB ID: 1Y6A) was downloaded from the Protein Data Bank. Water molecules and non-target protein components were removed, and the native ligand was separated using Discovery Studio 2017. Using AutoDockTools, polar hydrogens and Kollman charges were applied, and the structure was saved in .pdbqt format. The prepared protein structure is shown in Figure 1.

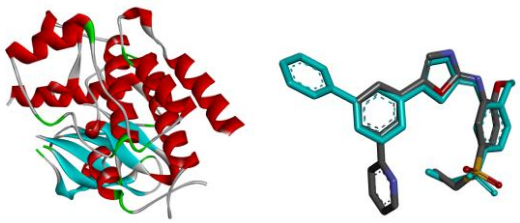

Figure 1. Structure of the prepared VEGFR2 target receptor and native ligand[12]

Ligand Preparation Results

Ligand preparation was performed by downloading the emodin structure data (PubChem CID: 3220) in .sdf format from PubChem. The file was opened using MarvinView and saved in .pdb format. The data in .pdb format was processed using AutoDockTools to check for rotatable bonds, then saved in .pdbqt format. The prepared structure is shown in **Figure 2**.

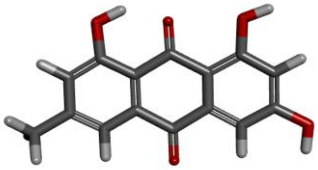

Figure 2. Structure of the prepared emodin ligand[13, 14]

Target Receptor Redocking Results

Redocking was performed to validate the docking protocol and ensure that the search parameters and scoring function accurately reproduce the known binding orientations within the VEGFR2 active site. The established binding pocket was defined using the grid box coordinates presented below, which served as the standardized docking environment for both the native ligand and axitinib.

Table 3. Coordinate Value and Grid Box Sizes

Parameter	X	y	z
Center (Å)	−0.333	30.358	16.864
Dimension (Å)	28	38	38

Redocking of the native crystallographic ligand produced an RMSD of 0.521 Å (**Table 4**), demonstrating that the docked pose closely overlapped with the experimentally resolved structure. This confirms that the defined grid box effectively captures the active site geometry and that the docking algorithm can reliably reproduce the essential ligand–receptor interactions present in the crystal complex.

Table 4. RMSD Values for Redocking VEGFR2 and Native Ligand

Receptor-Native Ligand Complex	RMSD
VEGFR2 dan 2-anilino-5-aryl-oxazole inhibitor	0.521

Note : RMSD (Root Mean Square Deviation)

To strengthen validation, axitinib, a clinically established VEGFR2 inhibitor, was also re-docked. The RMSD of 1.33 Å (**Table 5**) confirmed proper reproduction of its canonical binding orientation.

Table 5. RMSD Values for Redocking VEGFR2 and Its Known Inhibitor

Receptor-Native Ligand Complex	RMSD
VEGFR2 and axitinib	1.33

Note : RMSD (Root Mean Square Deviation)

These outcomes confirm that the docking protocol captures the VEGFR2 active-site environment with high fidelity and is reliable for subsequent ligand evaluations.

Results of Target Receptor and Ligand Docking

Molecular docking between the receptor and potential ligands was performed using AutoDockTools with AutoDock4. The gridbox parameters were adjusted based on the redocking results. The docking results are the binding energy values of the target receptor and ligands shown in **Table 6**.

Table 6. Binding Energy from Docking Results

Receptor-Ligand Complex	Binding Energy (kcal/mol)
VEGFR2 - Emodin	-7,78
VEGFR2 - Native Ligand	-4.81
VEGFR2 - Axitinib	-6.45

Based on the binding energy between the ligand and its target receptor, it was found that VEGFR2 shows a fairly strong interaction. Analysis and visualization of the docking interaction results are presented in the following figure.

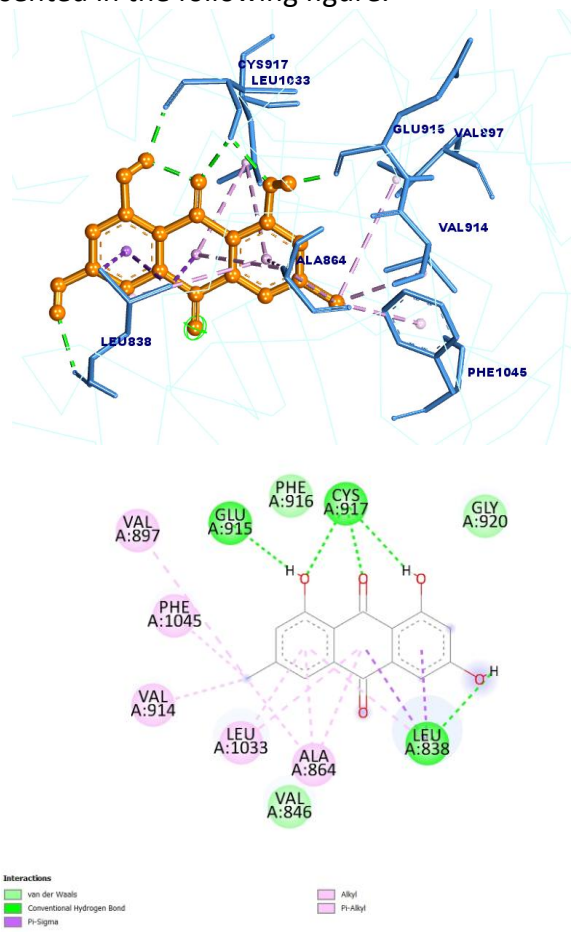

Figure 3. 2D and 3D visualization of molecular interactions between emodin and VEGFR2[12]

Discussions

Druglikeness of Potential Compuond

Emodin is a compound with a water solubility value (LogS) of -3.67 according to the SwissADME website. This value is categorized as slightly soluble.[11] This indicates that this compound is slightly soluble in water and may require appropriate drug formulation or administration route to increase its solubility and bioavailability.[15]

According to Lipinski's Rule of Five (RO5), emodin does not violate any criteria, indicating that this compound has good solubility and permeability.[16] This is consistent with the findings of Wasim et al. (2022), who also stated that emodin does not violate RO5, thus potentially having good oral bioavailability.[17]

Bevacizumab, an FDA-approved anti-angiogenic therapy for cervical cancer, can only be administered intravenously due to its large molecular weight (149 kDa) and poor oral bioavailability.[5] In contrast, emodin has good oral bioavailability and can be administered orally. This method provides greater comfort for patients because it is non-invasive, easy to perform, and allows patients to continue their daily activities without having to go to a medical facility.[18]

Redocking of Target Receptors and Native Ligands

Redocking was performed to validate the docking protocol and ensure that the defined binding site and scoring function reproduced established ligand orientations within the VEGFR2 active site.[19] The native crystallographic ligand served as the primary validation benchmark. Redocking yielded an RMSD of 0.521 Å and a binding energy of −4.81 kcal/mol, indicating excellent structural reproducibility and confirming that the grid box accurately captured the functional binding pocket.

To strengthen validation, axitinib, a clinically established VEGFR2 inhibitor, was also subjected to redocking. The procedure reproduced its reference pose with an RMSD of 1.33 Å and a binding energy of −6.45 kcal/mol, values consistent with typical AutoDock-derived profiles for tyrosine kinase inhibitors. The successful recovery of both ligand poses demonstrates that the docking parameters are reliable for predicting VEGFR2–ligand interactions.

When compared with both validated controls, emodin exhibits a more favorable binding energy (−7.78 kcal/mol). Its interaction energy is more negative than axitinib and significantly stronger than the native ligand, indicating a higher predicted affinity within the same validated docking framework.[20] This comparative performance further supports the credibility of emodin’s binding prediction and reinforces its potential role as a VEGFR2-targeting anti-angiogenic candidate.

Docking of Target Receptors and Ligands

Emodin exhibits a binding energy of −7.78 kcal/mol toward VEGFR2, indicating a stable and energetically favorable interaction, consistent with high-affinity ligand engagement.[19] This value is more negative than the emodin complex with SARS-CoV-2 MPro (-7.65 kcal/mol) and PRV protein (-5.66 and -5.25 kcal/mol).[21,22] Emodin also shows stable binding with the Caspase-3, Bcl-2, TRAF2, and other receptors in previous studies.[17] These comparisons suggest that emodin is structurally versatile in engaging protein pockets critical for disease-relevant pathways.

The emodin–VEGFR2 complex forms three hydrogen bonds and three van der Waals contacts, anchored around residues GLU915, CYS917, and LEU838. These residues are not arbitrary; they lie within the hinge region and ATP-binding cleft of the VEGFR2 kinase domain, a locus essential for receptor autophosphorylation and downstream signaling.[23,24] GLU915 is located within the conserved hinge backbone of VEGFR2, a region essential for anchoring ligands and establishing the hydrogen-bonding network required for ATP binding. When a compound interacts with this residue, it disrupts the canonical ATP-stabilizing framework, thereby hindering the receptor's ability to correctly position ATP for catalysis. CYS917 lies adjacent to this hinge segment and helps shape the entrance of the ATP pocket; ligand engagement at this point often forces the kinase into conformations that are incompatible with optimal catalytic activity. Meanwhile, LEU838 forms part of the hydrophobic interior of the binding pocket, providing nonpolar contacts that reinforce ligand stabilization. Interactions with this residue strengthen the overall binding pose through van der Waals forces, contributing to the persistence of inhibitor occupation within the ATP site.

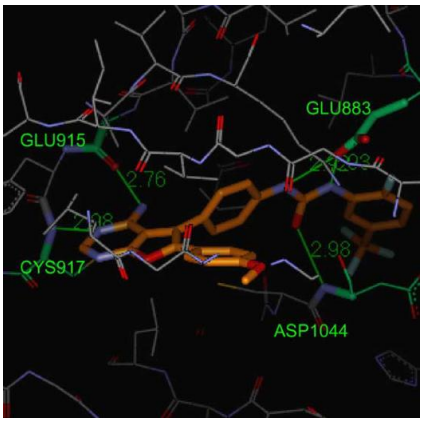

Figure 4. 3D visualization of molecular interactions between –NHCONH-(2-fluoro-5-trifluorophenyl) and VEGFR2[23]

These mechanistic roles explain why binding at these positions is likely to impede VEGFR2 kinase activity. The fact that axitinib also engages the hinge region and interacts with GLU915/CYS917, as shown by Miyazaki et al., further validates the functional relevance of emodin's binding mode.[25] Axitinib is a second-generation tyrosine kinase inhibitor that selectively inhibits VEGFR-1, VEGFR-2, and VEGFR-3, effectively inhibiting angiogenesis, tumor growth, and metastasis with 50-450 times higher potency than first-generation VEGFR inhibitors.[26] This suggests that emodin could be a potential compound for overcoming bevacizumab resistance because emodin binds to VEGFR2 followed by binding to other receptors that can help inhibit tumor angiogenesis.[17]

The stronger binding energy of emodin relative to axitinib in this study's docking protocol suggests a tight theoretical interaction, but this should be interpreted within the limitations of docking scores.[27] Even so, the consistent inhibition of hinge-binding inhibitors strengthens the hypothesis that emodin may hinder VEGFR2-driven angiogenesis through direct catalytic interference. This also provides a mechanistic rationale for exploring emodin as a candidate for overcoming bevacizumab resistance, given its potential to target VEGFR2 while concurrently modulating other angiogenesis-related pathways through its previously reported interactions with multiple signaling proteins.

Because AutoDock4 operates using a stochastic search algorithm, pose variation between runs is expected.[28] This noise can be minimized through repeated docking and convergence assessment, helping ensure that the identified emodin–VEGFR2 pose represents a stable and reproducible interaction.

Conclusion

Based on the analysis of the research results conducted using computational methods, it can be concluded that :

1. There is an interaction between the bioactive compound emodin and VEGFR2 that forms 3 hydrogen bonds and 3 van der Waals bonds.
2. The best binding pose interacts with amino acids GLU915, CYS 917, and LEU838 as hydrogen bonds and PHE916, GLY920, and VAL846 as van der Waals interactions.
3. The interaction formed between emodin and VEGFR2 produces a binding energy of -7.78 kcal/mol from the best binding pose.

These findings position emodin as a promising scaffold for the development of VEGFR2-targeted anti-angiogenic therapies. The strong binding affinity, engagement of catalytically relevant hinge residues, and favorable ADMET predictions justify further investigation through molecular dynamics simulations, kinase inhibition assays, and structural optimization to enhance potency and selectivity. Integration with in vitro angiogenesis models and in vivo tumor-growth studies will be essential to validate its therapeutic potential and assess its capacity to overcome resistance observed with current VEGFR2 inhibitors.

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