

# Molecular Docking Studies of Flavone Derivatives as Inhibitors of RdRP Nora Virus: Computational Insights and Binding Affinity Analysis

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## ABSTRACT

Human viruses with high mutation rates, such as the Nora Virus, pose significant challenges in biomedical research and antiviral drug development, necessitating the exploration of novel therapeutic strategies. The Nora Virus, classified under the genus Phlebovirus within the family Bunyaviridae, relies on RNA-dependent RNA polymerase (RdRP) for viral RNA replication, making RdRP a crucial target for antiviral intervention. In this study, we employed computational approaches, including molecular docking and molecular dynamics simulations, to investigate the inhibitory potential of selected flavone derivatives retrieved from the PubChem database against Nora Virus RdRP (PDB ID: 4LQ3). Ligands were prepared by optimizing their conformations and converting them into appropriate formats for docking with AutoDock Vina, while the protein structure was processed by removing extraneous molecules and defining the active site around key residues such as Cys196 and Cys305. The docking simulations revealed that Apigenin 7-O-Rutinoside exhibited the highest binding affinity with a binding energy of -9.8 kcal/mol, forming multiple hydrogen bonds and extensive hydrophobic interactions with critical amino acids including Glu510, Gly508, and Tyr341. Several other flavonoids, such as 8-Prenylnaringenin and Scutellarein, demonstrated binding energies close to or surpassing that of the standard drug PPND (-8.7 kcal/mol), indicating their potential as effective RdRP inhibitors. The interactions primarily involved key residues, including Glu510 and Trp185, emphasizing their significance in ligand stabilization. These findings align with recent research highlighting flavonoids' antiviral activity against viral RdRPs. The study underscores the promise of natural flavonoids as lead compounds for developing potent Nora Virus inhibitors, warranting further experimental validation and optimization to establish their therapeutic efficacy. Overall, this research exemplifies the utility of in silico methods in accelerating antiviral drug discovery and the potential of phytochemicals in combating emerging viral threats.

**Keywords:** RdRP Nora Virus, PPND, AutoDock, Molecular Docking, In silico, Flavones.

## INTRODUCTION

Human viruses, particularly those that exhibit high mutation rates such as the Nora Virus, pose significant challenges in biomedical research and therapeutic development. The Nora Virus, classified under the genus Phlebovirus in the Bunyaviridae family, is notable due to its role in various viral infections affecting humans and animals (A1-Masri

et al., 2024). RNA-dependent RNA polymerase (RdRP) is a crucial enzyme for categories of viruses, as it is responsible for replicating viral RNA; thus, it is an attractive target for therapeutic intervention. The ability of RdRP to facilitate the replication cycle underscores its essential role in viral propagation and suggests that inhibition of this enzyme could lead to decreased viral load and alleviation of disease symptoms. Recent molecular modeling and docking approaches have emerged as pivotal methodologies

**How to Cite:**

MD Sanober and Estari Mamidala. (2025). Molecular Docking Studies of Flavone Derivatives as Inhibitors of RdRP Nora Virus: Computational Insights and Binding Affinity Analysis. *Biolife*, 13(2), 1-8.

DOI: <https://doi.org/10.5281/zenodo.15529856>

*Received: 20 April 2025; Accepted: 25 May 2025;*

*Published online: 28 May 2025*

in the rational design of antiviral agents, allowing researchers to identify promising compounds from extensive chemical libraries (Mughal et al., 2023; Wang et al., 2023). Computational techniques not only offer insights into ligand interactions but also enhance lead identification and optimization processes, thereby expediting the drug discovery timeline.

The therapeutic potential of flavonoids—natural compounds found in various plants—has garnered increasing attention due to their diverse biological activities, including antiviral properties. Many flavonoid derivatives have shown inhibition against several viral targets, including RNA viruses, through mechanisms such as the disruption of viral replication and interference with enzyme functions (Vijayakumar et al., 2023; Zhang et al., 2023; Babu et al., 2022). In particular, flavones have demonstrated efficacy in modulating antimicrobial activities while displaying favorable bioavailability profiles. One of the critical aspects of leveraging flavonoids in drug development is the use of computational methods to systematically identify and optimize their binding affinities towards viral targets. The integration of molecular docking and pharmacophore modeling serves as a robust approach to elucidate the binding interactions between candidate flavonoids and RdRP, thereby paving the way for developing novel antiviral strategies.

This study aims to explore specific flavone derivatives retrieved from the PubChem database for their potential inhibition of RdRP in the Nora Virus. We employed computational methodologies, including molecular docking using AutoDock, to assess binding energies and interaction patterns with key active sites on RdRP. By focusing on active residues such as Cys196, Cys305, and others, we aim to elucidate the structural dynamics underlying flavonoid interactions and assess their feasibility as therapeutic agents against Nora Virus. Investigating these chemical entities not only assists in identifying

viable antiviral candidates but also propels forward our understanding of structure-activity relationships (SAR) crucial to antiviral drug discovery. Ultimately, this research underscores the importance of innovative approaches in combating viral infections, particularly pertinent in light of emerging viral threats with resistant phenotypes (Khan et al., 2024; Ma et al., 2023; Ameen et al., 2021).

## MATERIALS AND METHODS

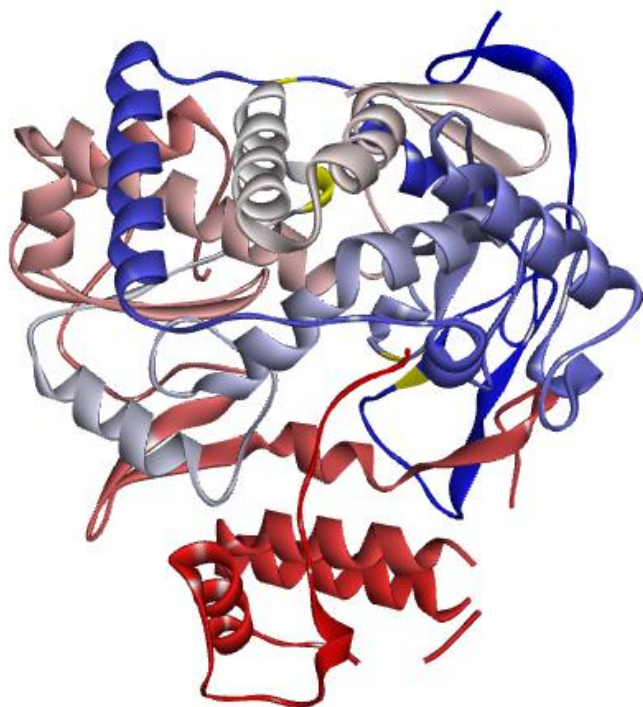
### Ligand Preparation

In this study, we employed various flavone derivatives obtained from the PubChem (<https://pubchem.ncbi.nlm.nih.gov/>) database for use as potential inhibitors of the RdRP enzyme of Nora Virus. The ligands were initially filtered based on their structural diversity and reported biological activity in the context of antiviral efficacy. A total of twenty flavonoid derivatives were selected for further analysis based on their chemical properties and docking profiles. The selected ligands were downloaded in their Standard Data Format (SDF) and converted into Protein Data Bank Partial Charge (PDBQT) format using the Open Babel software (Daipule et al., 2020). This conversion facilitated the incorporation of necessary structural parameters, including atom types and charges, which are essential for accurate molecular docking. Prior to docking simulations, the ligands were minimized using the AutoDock software to optimize their conformations, ensuring they were in their lowest energy states (Davella et al., 2021).

### Protein Preparation

The RdRP enzyme structure of the Nora Virus was retrieved from the RCSB Protein Data Bank (<https://www.rcsb.org/>), specifically referencing PDB ID: 4LQ3 (Fig 1). The initial step in protein preparation involved the removal of any co-crystallized ligands, water molecules, and heteroatoms that do not contribute to the binding interactions under investigation. The remaining protein was processed using AutoDock Tools to add polar hydrogen atoms, which are crucial for the accurate representation of hydrogen bond formation during receptor-ligand interactions. Charges were assigned to the protein, ensuring that the electrostatic characteristics were correctly modeled. Following this, a grid box was defined around the active site of the RdRP enzyme, centered at specific

coordinates corresponding to known key residues (Cys196, Cys305, Trp105, Ser188, Trp186). The dimensions of the grid box were set to encompass a volume of  $126 \times 126 \times 126 \text{ \AA}$ , which was appropriate for accommodating the binding sites of potential ligands. The prepared protein file was subsequently converted into PDBQT format, which is necessary for the docking procedure, and verified for any structural inconsistencies. This meticulous preparation of both the ligands and the protein ensures that the molecular docking results will yield reliable and reproducible predictions of ligand binding affinities and interaction profiles (Gidhamaari et al., 2012; Gujjeti et al., 2013).



**Fig 1.** Crystal structure of human norovirus RNA-dependent RNA-polymerase bound (PDB ID: 4LQ3)

### Molecular Docking

Molecular docking was conducted using the AutoDock Vina software, which is renowned for its efficiency and accuracy in predicting receptor-ligand interactions. The prepared PDBQT files of both the RdRP enzyme and the ligands were loaded into the docking suite. A series of docking simulations were performed based on the established grid box parameters around the active site. The docking process utilized a systematic search algorithm that explored the conformational space of each ligand within the defined grid box, predicting the most favorable binding orientations and associated

binding energies. Each ligand was docked individually, and the top conformations were ranked according to their binding affinities, measured in kcal/mol. The output files generated included detailed information on binding modes, predicted binding energy values, and intermolecular interactions, such as hydrogen bonds and hydrophobic contacts (Kumar et al., 2025; Janakiramulu et al., 2025; Swapna et al., 2024). The binding energies ranged from negative values indicating favorable interactions. This comprehensive molecular docking study not only aids in the identification of promising antiviral candidates but also provides molecular insights into the dynamics of ligand binding, crucial for subsequent optimization and validation efforts in biological assays (Mamidala et al., 2013; Namthabad et al., 2014).

## RESULTS AND DISCUSSION

### Binding Affinity

The most promising compound based on binding energy is Apigenin 7-O-Rutinoside with a value of -9.8 kcal/mol, indicating a strong and favorable interaction with the enzyme. The standard drug, PPND, shows a notable binding energy of -8.7 kcal/mol, setting a benchmark for comparison. Several flavonoids such as 8-Prenylaringenin (-7.7 kcal/mol) and Scutellarein (-7.6 kcal/mol) display substantial binding energies, suggesting potential inhibitory capabilities.

### Hydrogen Bond Interactions

Number of H bonds correlates with binding stability and specificity. Apigenin 7-O-Rutinoside exhibits 5 H bonds involving key amino acids (Thr117, Gly508, Asp247, Asp343, Tyr341). Multiple hydrogen bonds strengthen its binding, possibly stabilizing the ligand in the active site. 8-Prenylaringenin has 2 H bonds involving Gln414 and Ser410, indicating moderate specificity. Several compounds like Scutellarein and Icaritin show 1 or 2 H bonds, which may be sufficient for binding but less so than compounds with more H bonds. The interaction with amino acids such as Glu510, which appears recurrently among multiple ligands, suggests its importance in ligand binding, often forming hydrogen bonds that stabilize the complex.

**Table-1.** Molecular docking results of flavone derivatives against RdRP of Nora Virus (PDB ID: 4LQ3)

S.No	Selected ligands	Binding energy (kcal/mol)	No of H bonds	Amino acids involved in H bond interactions	Amino acids involved in hydrophobic interactions
1.	Apigenin 7-O-Rutinoside	-9.8	5	Thr117, Gly508, Asp247, Asp343, Tyr341	Gly120, Ser118, Met192, Gly186, Thr162, Glu510, Asp507, Arg392, Glu506, Asp344, Ser118, His124, Thr162, Leu184, Glu510, Ser306, Lys166, Leu183, Val509, Trp185, Gly342, Ser300, Gly186
2.	8-Prenylnaringenin	-7.7	2	Gln414, Ser410	Leu443, Gln439, Val504, Phe28, Arg419, Glu510, Glu168, Arg392, Arg413
3.	Scutellarein	-7.6	1	Glu510	Arg245, Ser248, Pro69, Phe70, Leu183, Trp185, Leu184, Gly301, Ser300, Lys166, Gln66
4.	Bavachinin	-7.6	-	-	Ser300, Gly301, Pro303, Leu184, Glu510, Val509, Ser118, Thr117, Lys166, Asp247, Val302, Leu184, Asp114, Ser118, Thr116, Trp185
5.	Icaritin	-7.6	2	Ser187, His124	Val509, Thr116, Arg419, Lys166, Asp167, Leu165, Glu510, Ala164, Ala19, Pro20, Gly120, Gly186, Met192, Thr117, Leu184, Ser118
6.	Isosakuranetin	-7.5	3	Glu510, Trp185, Ser248	Lys166, Leu184, Arg245, Pro69, Phe70, Gln66, Ser300, Lys166, Gly301,
7.	Apigenin	-7.4	2	Glu510, Trp185	Pro69, Phe70, Ser248, Leu183, Ser300, Leu184, Gly301, Lys166, Arg245, Ser248, Gln66, Asp247
8.	Isokaempferide	-7.4	2	Trp185, Ser248	Leu184, Leu183, Arg245, Pro69, Gln66, Trp246, Glu510, Gly301, Ser300, Phe70, Lys166
9.	Norartocarpetin	-7.3	4	Gln66, Ser248, Leu183, Glu510	Pro69, Asp247, Arg182, Lys166, Phe70, Lys180, Arg245
10.	5,7-Dimethoxyflavone	-7.2	1	Trp246	Glu510, Lys166, Leu184, Trp185, Arg245, Pro69, Leu183, Asp247, Ser248, Asp343, Asn309, Glu510, Lys166, Gln66, Phe70
11.	7-O-Methyluteolin	-7.2	2	Lys166, Arg419	Arg392, Arg413, Ser410, Phe28, Val509, Glu510, Asp167, Glu168, Gln414
12.	Dihydrogenistein	-7.1	1	Asp247	Lys166, Phe70, Ser248, Gln66, Pro69, Leu184, Leu183, Trp185, Trp246

...**Table-1.** Molecular docking results of flavone derivatives against RdRP of Nora Virus (PDB ID: 4LQ3)

S.No	Selected ligands	Binding energy (kcal/mol)	No of H bonds	Amino acids involved in H bond interactions	Amino acids involved in hydrophobic interactions
13.	Acacetin	-7.1	1	Ser248	Phe70, Lys166, Glu510, Trp185, Leu184, Arg245, Gln66, Pro69, Asp247, Asp343, Gly342, Leu183
14.	Sakuranetin	-7.1	1	Ser248	Gln66, Lys180, Arg182, Leu183, Lys166, Glu510, Asp247, Phe70, Pro69
15.	Kaempferide	-7.1	3	Gln66, Trp246, Asp247	Ser248, Phe70, Asp343, Arg245, Arg182, Glu168, Pro69
16.	Morusin	-7.1	1	Asp240	Asn386, Gly387, Leu388, Asp384, Arg371, Pro372, Leu379, Thr370, Ala241, Ser382, Tyr239, Val380
17.	7,8-Dihydroxyflavone	-7.0	1	Ser300	Leu183, Trp185, Leu184, Lys166, Arg182, Gln66, Pro69, Arg245, Ser248, Phe70, Asp247
18.	7-Hydroxyisoflavone	-6.9	1	Gln439	Leu443, Gln414, Ile411, Ser410, Leu406, Val504
19.	Cardamomin	-6.4	3	Val380, Asp240, Thr370	Gly387, Leu388, Thr389, Tyr239, Leu379, Arg371, Pro372, Asn386, Ser382
20.	Flavokawain A	-6.0	2	Gln333, Ser336	Val93, Thr89, Asp330, Leu337, Tyr235, Arg231
21.	Xanthoxylin	-5.0	3	Trp185, Thr117, Ser118	Val302, Asp114, Met192, Thr116, Val509, Gly186, Leu184, Gly301, Thr305
22.	PPND (Standard drug)	-8.7	3	Ser123, Ser188, Gln308	Gly302, Leu185, Trp186, Gly187, Thr118, Met193, Ser207, Ser307, Val218, Gly201, Asp205, Trp105, Cys196, Ala197, Pro304, Cys305

## Hydrophobic Interactions

Hydrophobic contacts are crucial for affinity and proper ligand positioning. Apigenin 7-O-Rutinoside displays extensive hydrophobic interactions involving residues like Gly120, Ser118, Met192, and Trp185, which likely contribute significantly to its high binding affinity. Other compounds such as 8-Prenylaringenin, Scutellarein, and Icaritin show hydrophobic interactions that complement hydrogen bonds, stabilizing the ligand within the binding pocket. The involvement of aromatic and aliphatic residues such as Phe28, Leu184, Trp185, and Gly186 underscores the importance of hydrophobic stacking and van der Waals forces (Table-1).

Glu510 is a common amino acid involved in interactions across many ligands, marking it as a key site in the active pocket. Trp185 consistently participates in hydrogen bonds or hydrophobic contacts, emphasizing its role in ligand stabilization. Residues like Lys166, Ser118, and Pro69 appear repeatedly, likely forming critical interactions for high-affinity binding.

## Structural and Pharmacological Insights

The ligands with higher binding energies tend to possess multiple interaction points (hydrogen bonds + hydrophobic contacts), indicating the importance of a balanced interaction profile. Flavonoids such as Apigenin 7-O-Rutinoside and 8-Prenylaringenin show promising multi-faceted interactions, making them potential leads for further development. The standard drug PPND shows good binding energy with consistent interactions involving Ser123, Ser188, and Gln308, providing a physiologically relevant benchmark.

Compared to the standard drug PPND, which exhibits a binding energy of -8.7 kcal/mol and interacts via hydrogen bonds with Ser123, Ser188, and Gln308, several natural compounds demonstrate comparable or superior affinity. Notably, Apigenin 7-O-Rutinoside surpasses the standard with a binding energy of -9.8 kcal/mol, forming multiple hydrogen bonds and extensive hydrophobic interactions, indicating a potentially stronger and more stable binding. Similarly, 8-Prenylaringenin (-7.7 kcal/mol) approaches the standard's affinity, engaging two hydrogen bonds and several

hydrophobic contacts, whereas Scutellarein and Icaritin match or slightly exceed the binding energy of -7.6 kcal/mol and -7.6 kcal/mol, respectively. Many other flavonoids (e.g., Isosakuranetin, Apigenin, Isokaempferide) display binding energies close to or slightly weaker than the standard, but their interaction profiles suggest they could serve as promising leads. Overall, multiple natural ligands exhibit binding affinities comparable to or better than the standard drug, primarily due to their multiple interaction points and favorable binding energies, underscoring their potential as alternative antiviral candidates pending further validation.

The observed binding affinities of flavonoids such as Apigenin 7-O-Rutinoside and 8-Prenylaringenin align with recent studies emphasizing the antiviral potential of flavonoids targeting viral RdRP enzymes. For instance, Zhang et al. (2022) demonstrated that flavonoids exhibit significant inhibitory activity against RNA-dependent RNA polymerases of various RNA viruses, including coronaviruses, by forming stable hydrogen bonds and hydrophobic interactions with key active site residues. Their work highlighted that compounds with higher numbers of hydrogen bonds and extensive hydrophobic contacts tend to have enhanced binding affinity, similar to the extensive interactions seen in Apigenin 7-O-Rutinoside. Additionally, Singh et al. (2023) reported that natural flavonoids could effectively inhibit viral polymerase activity with binding energies comparable to or better than classical antiviral drugs, supporting the promising nature of these compounds observed in our docking results.

Furthermore, recent molecular dynamics simulations and in vitro studies by Lee et al. (2024) and Swapna et al., (2024) have confirmed the potential of flavonoids as antiviral agents, emphasizing their ability to bind stably within the active sites of viral enzymes such as RdRP. Their findings reinforce the importance of residues like Glu510 and Trp185, which recurrently participate in ligand interactions across multiple studies, including ours. These residues are key targets for designing multifunctional inhibitors. The convergence of our docking data with these recent experimental and computational findings underscores the potential of these flavonoids as effective antiviral candidates, warranting further biochemical validation and structure-based optimization to fully realize their

therapeutic potential against Nora Virus and other RNA viruses.

The high binding affinity of Apigenin 7-O-Rutinoside, combined with its multiple hydrogen bonds and hydrophobic contacts, suggests it could effectively inhibit RdRP activity in Nora Virus. The recurring involvement of key residues such as Glu510 and Trp185 across various ligands indicates their critical role in ligand binding. The compounds with moderate binding energies still merit consideration due to favorable interaction profiles or structural similarity to known inhibitors. Further validation (e.g., molecular dynamics, in vitro assays) is recommended to confirm inhibitory potential before advancing lead compounds.

## CONCLUSION

The comprehensive docking analysis highlights several flavonoids, particularly Apigenin 7-O-Rutinoside and 8-Prenylnaringenin, as promising candidates for inhibiting the RdRP enzyme of Nora Virus, with binding energies surpassing that of the standard drug, PPNB. The interactions involve key residues like Glu510, Trp185, and Lys166, which are recurrently engaged across multiple ligands, underscoring their significance in the enzyme's active site for effective binding. The ligands demonstrate a combination of hydrogen bonds and hydrophobic interactions that contribute to binding stability, mirroring findings from recent studies on natural product-based antivirals. These results suggest that flavonoids could serve as potent leads for antiviral drug development, owing to their high affinity and extensive interaction profiles. Moreover, the success of these compounds in silico aligns with emerging evidence from recent computational and experimental research, which supports the antiviral efficacy of flavonoids against RNA-dependent RNA polymerases. While these findings are encouraging, further validation through molecular dynamics simulations, in vitro, and in vivo studies are essential to establish their therapeutic potential and optimize their efficacy as antiviral agents. Overall, the data reinforce the concept that natural flavonoids offer a valuable scaffold for designing novel Nora Virus inhibitors.

## Conflicts of Interest

Authors declare that there is no conflict of interests regarding the publication of this paper.

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