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In Silico Evaluation of 3,4,5-Tri-O-Galloylquinic Acid Against Chandipura Virus Glycoprotein G Using Molecular Docking, Swiss ADME, and Toxicity Prediction

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Abstract

Background: Chandipura virus (CHPV) is an emerging neurotropic pathogen responsible for acute encephalitis syndrome with high mortality, particularly among children. In the absence of a specific antiviral therapy, the identification of novel inhibitors targeting the viral glycoprotein G is of considerable interest.

Methods: The antiviral potential of 3,4,5-tri-O-galloylquinic acid against Chandipura virus glycoprotein G was evaluated using molecular docking, with favipiravir as the reference antiviral. Protein–ligand interactions were analyzed using Discovery Studio Visualizer. Pharmacokinetic properties, drug-likeness, and blood–brain barrier permeability were predicted using Swiss ADME and the Boiled Egg model, while the toxicity profile was assessed using the ProTox-3.0 web server.

Results: 3,4,5-tri-O-galloylquinic acid exhibited stronger binding affinities than favipiravir, with docking scores of −4.7 and −6.0 kcal/mol against glycoprotein G structures 4D6W and 5MDM, respectively, compared with −3.4 and −4.0 kcal/mol for favipiravir. The phytochemical formed multiple hydrogen bonds and hydrophobic interactions, indicating stable protein–ligand complexes. Swiss ADME predicted low gastrointestinal absorption and poor oral bioavailability for the phytochemical, whereas favipiravir exhibited more favorable pharmacokinetic properties. The Boiled-Egg model predicted that neither compound is likely to cross the blood–brain barrier by passive diffusion. ProTox-3.0 predicted low acute oral toxicity (LD_{50} = 3700 mg/kg, Toxicity Class 5) with no predicted hepatotoxicity, carcinogenicity, mutagenicity, or cardiotoxicity.

Conclusion: 3,4,5-tri-O-galloylquinic acid demonstrated promising binding affinity and a favorable predicted safety profile, supporting its potential as a lead compound against CHPV. However, optimization of its pharmacokinetic properties and experimental validation are required before therapeutic application.

Keywords: Chandipura virus; Glycoprotein G; Molecular docking; 3,4,5-tri-O-galloylquinic acid; Swiss ADME; ProTox-3.0

Introduction

Chandipura virus (CHPV) is an emerging zoonotic pathogen that poses a significant public health threat, particularly in the Indian subcontinent. The virus was first isolated in 1965 during an outbreak of dengue-like febrile illness in Chandipur village, Maharashtra, India [1]. CHPV belongs to the family Rhabdoviridae and is characterized as a bullet-shaped, enveloped virus containing a single-stranded, negative-sense RNA genome [2]. Infection with CHPV is associated with rapidly progressive acute encephalitis, predominantly affecting children younger than 15 years of age, with reported case fatality rates ranging from 57% to 70% during outbreaks. Clinical manifestations include high fever, vomiting, seizures, altered consciousness, and coma, often resulting in death within 48 hours of hospital admission in severe cases. The virus is primarily transmitted by the bites of sand flies of the genera Phlebotomus and Sergentomyia and has also been reported to be transmitted by the mosquito Aedes aegypti [3]. The latest documented outbreak occurred between June and August 2024 in multiple districts of Gujarat, India, highlighting the continued public health importance of this emerging viral disease [4].

Currently, no specific antiviral therapy has been approved for the treatment of Chandipura virus (CHPV) infection, and patient management is primarily supportive. Clinical treatment focuses on alleviating symptoms and preventing complications, with agents such as mannitol and furosemide being administered to reduce cerebral edema and control elevated intracranial pressure. Likewise, no licensed vaccine is currently available for the prevention of CHPV infection. Given the absence of approved antiviral drugs or vaccines, the development and identification of effective antiviral agents remain a priority [5]. Drug repurposing has emerged as a promising strategy, as existing antiviral compounds with established safety profiles may offer a faster and more cost-effective approach for identifying potential therapeutic options against CHPV by inhibiting viral replication. The viral glycoprotein (G) is an essential structural protein responsible for viral attachment, receptor recognition, and membrane fusion during host-cell entry [6]. Owing to its indispensable role in viral infectivity, the G glycoprotein represents an attractive molecular target for structure-based antiviral drug discovery. Recent advances in structural biology have provided experimentally resolved three-dimensional structures of the CHPV G glycoprotein, enabling computational approaches such as molecular docking to identify potential antiviral compounds that may interfere with virus-host interactions.

Natural polyphenolic compounds have gained increasing attention as potential antiviral agents due to their diverse pharmacological activities and ability to modulate multiple viral and host targets. Among these, 3,4,5-tri-O-galloylquinic acid (GQA), a galloylated quinic acid derivative isolated from medicinal plants, has demonstrated antiviral activity through inhibition of HIV-1 and Moloney murine leukemia virus (M-MLV) reverse transcriptases, suggesting its potential as a broad-spectrum antiviral phytochemical [7]. Furthermore, galloylquinic acid derivatives have shown promising in silico interactions with SARS-CoV-2 Spike and RNA-dependent RNA polymerase (RdRp) proteins, supporting their utility as lead molecules for antiviral drug discovery.

Despite these encouraging findings, no computational or experimental studies have investigated the interaction of 3,4,5-tri-O-galloylquinic acid with the Chandipura virus G glycoprotein. Therefore, the present study aimed to evaluate the binding potential of 3,4,5-tri-O-galloylquinic acid against two experimentally determined CHPV G glycoprotein structures and [8,9] using molecular docking and protein–ligand interaction analysis. Favipiravir, a broad-spectrum antiviral agent, was employed as a reference compound for comparative evaluation [10]. The findings of this study may provide preliminary insights into the potential of galloylquinic acid derivatives as lead molecules for the development of therapeutic interventions against CHPV.

Methodology

Molecular docking

The three-dimensional structures of the Chandipura Virus glycoprotein G (PDB ID: 4D6W and 5MDM) were selected from the Protein Data Bank (PDB), a digital archive of protein structural information. AutoDock Vina (version 1.2.7) [11] was used to perform molecular docking studies to assess interactions between these target proteins and 3,4,5-tri-O-galloylquinic acid ligands. Before docking, the protein and ligand files were prepared in PDBQT format. Preprocessing included removing water molecules and introducing polar hydrogen atoms and Kollman charges to ensure proper charge distribution and protein polarity. Grid preparation and receptor setup were carried out using AutoDockTools (ADT). The docking grid parameters, including grid center coordinates and box dimensions, were selected to adequately cover the active binding regions of the respective target proteins. A grid spacing value of 0.375 *Å* was used during grid preparation. The docking was performed using an active-site docking approach rather than blind docking. In this procedure, the PDB structure of the target protein was first selected, and the co-crystallized ligand/drug was removed. The grid box was then generated around the binding region previously occupied by the native ligand. Therefore, the docking region was defined based on the co-crystallized ligand binding pocket of the target protein as follows

i. **4D6W:** Grid box size of 18 × 22 × 22 *Å* (x, y, z) and center coordinates at -37.58278571 × -24.01707143 × -35.60314286 *Å*.

ii. **5MDM:** Grid box size of 18 × 22 × 22 *Å* (x, y, z) with center coordinates at 77.98371429 × 89.65771429 × -0.176642857 *Å*.

The prepared protein structures in PDBQT format were then utilized for molecular docking to investigate potential binding affinities and interaction patterns between the ligands and target proteins. Molecular docking studies were conducted using the prepared protein structures in AutoDock Vina to assess ligand binding affinity and interactions with key target proteins. Docking was performed within an optimized grid box, generating ten poses for each ligand. The binding energy of 3,4,5-tri-O-galloylquinic acid and Ribavirin was evaluated. The docking interactions were visualized and analyzed using Biovia Discovery Studio 2024 for a detailed understanding of ligand-protein interactions.

Swiss ADME analysis

The pharmacokinetic properties, physicochemical characteristics, drug-likeness, medicinal chemistry parameters, and oral bioavailability of 3,4,5-tri-O-galloylquinic acid and the reference antiviral favipiravir were evaluated using the Swiss ADME web server (http://www.swissadme.ch). The canonical SMILES of each compound were retrieved from the PubChem database and submitted to the SwissADME server for analysis. The predicted parameters included molecular weight, topological polar surface area (TPSA), lipophilicity (Log P), water solubility (Log S), gastrointestinal (GI) absorption, blood-brain barrier (BBB) permeability, P-glycoprotein (P-gp) substrate prediction, skin permeation coefficient (Log Kp), drug-likeness according to Lipinski, Ghose, Veber, Egan, and Muegge rules, bioavailability score, PAINS and Brenk structural alerts, lead-likeness, and synthetic accessibility.

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The Boiled-Egg model implemented in Swiss ADME was used to predict passive gastrointestinal absorption and blood-brain barrier permeation based on lipophilicity and topological polar surface area (TPSA). The model also predicts whether compounds are substrates of P-glycoprotein, an important efflux transporter influencing oral drug absorption.

In silico toxicity prediction

The toxicity profile of 3,4,5-tri-O-galloylquinic acid was predicted using the ProTox-3.0 web server. The canonical SMILES of the compound was submitted to the server to estimate acute oral toxicity (LD_{50}), toxicity class, organ toxicity, toxicity endpoints, Tox21 pathway interactions, and cytochrome P450-mediated metabolism. The predictions were used to evaluate the compound's preliminary safety profile for potential therapeutic applications [12].

Results

Binding affinity of 3,4,5-tri-O-galloylquinic acid and favipiravir with 4D6W and 5MDM

The molecular docking analysis demonstrated that 3,4,5-tri-O-galloylquinic acid (GA) exhibited stronger binding affinity toward the Chandipura virus glycoprotein G than the reference antiviral favipiravir (FA). For the protein structure 4D6W, GA showed a binding affinity of −4.7kcal/mol, whereas FA exhibited a lower binding affinity of −3.4kcal/mol. Similarly, against the 5DMD glycoprotein G structure, GA achieved a binding affinity of −6.0kcal/mol, compared with −4.0kcal/mol for FA. GA consistently produced more negative binding energies than FA across both glycoprotein G structures, indicating a stronger predicted interaction with the target protein. Among all the docked complexes, the GA–5DMD complex demonstrated the highest binding affinity (−6.0kcal/mol), suggesting that this ligand formed the most stable protein–ligand complex in the docking simulations. In contrast, the FA–4D6W complex exhibited the weakest binding affinity (−3.4kcal/mol). These findings suggest that 3,4,5-tri-O-galloylquinic acid possesses a greater predicted binding potential toward the Chandipura virus glycoprotein G than favipiravir, supporting its potential as a promising lead compound for further investigation.

Favipiravir interacted with the Chandipura virus glycoprotein G (4D6W) through two conventional hydrogen bonds with Asn84 and Ser164, a halogen bond with Asn163, carbon-hydrogen bonds with His60 and His162, and van der Waals interactions with Thr103 and Ser165 Figure 1. 3,4,5-tri-O-galloylquinic acid exhibited a strong interaction with the Chandipura virus glycoprotein G (4D6W), forming eight conventional hydrogen bonds with Thr160, Ile161, His162, Lys86 (2 bonds), Asn84, and Asn163, along with π-alkyl/alkyl interactions involving Lys86. In addition, Asp159, Ser164, Ser165, Ile85, Pro87, and Thr88 contributed through van der Waals interactions, resulting in a stable protein–ligand complex Figure 2.

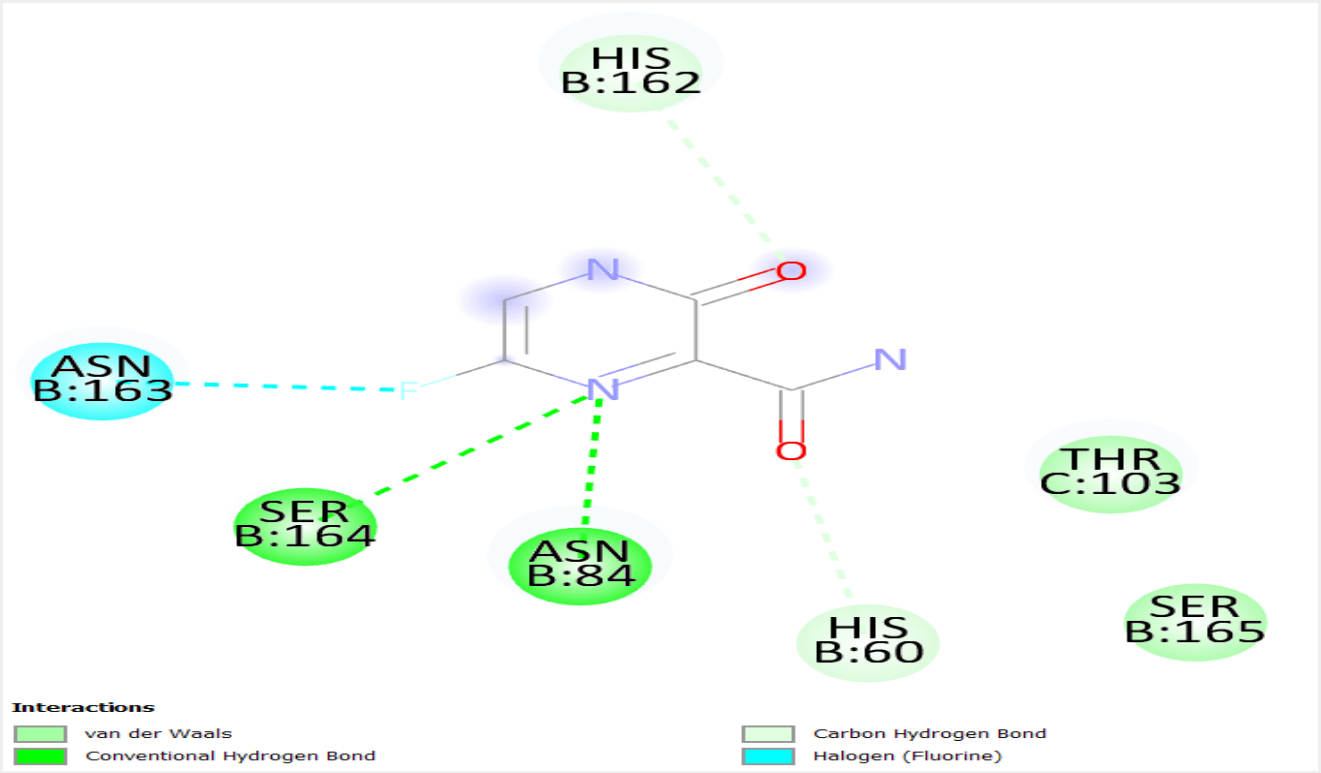

Figure 1: Two-dimensional interaction diagram of favipiravir docked with the Chandipura virus glycoprotein G (PDB ID: 4D6W).

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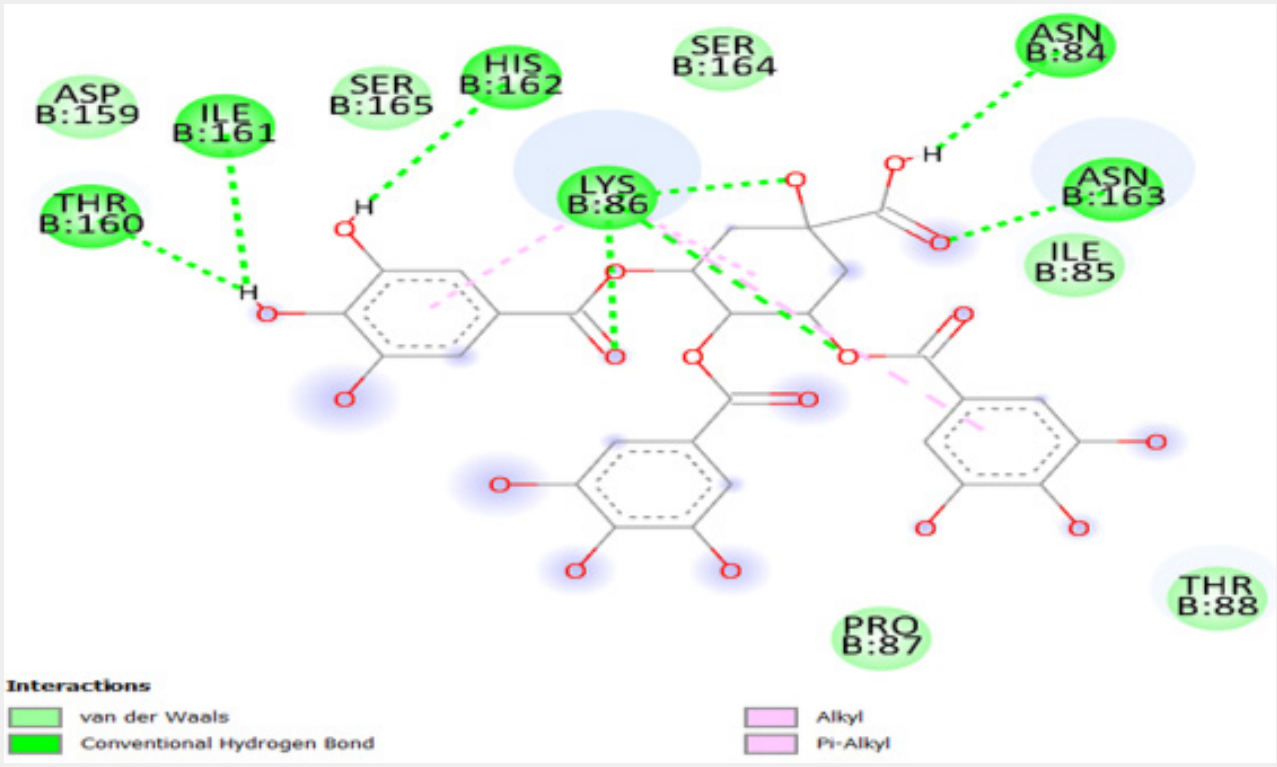

Figure 2: Two-dimensional interaction diagram of 3,4,5-tri-O-galloylquinic acid docked with the Chandipura virus glycoprotein G (PDB ID: 4D6W).

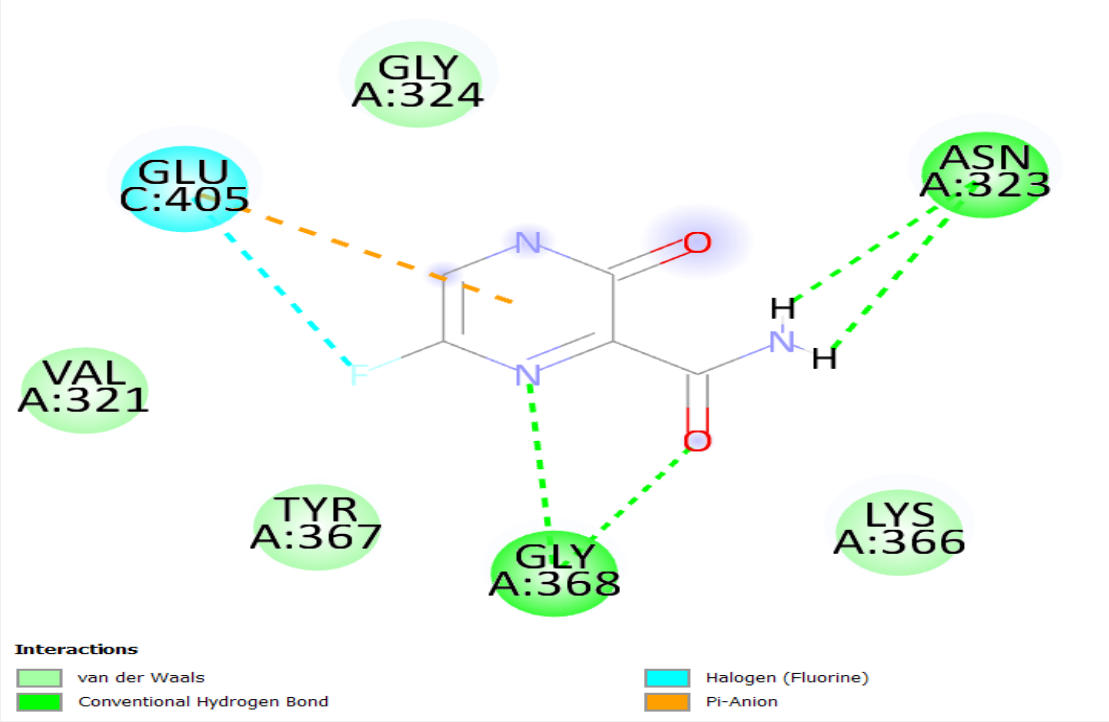

Figure 3: Two-dimensional interaction diagram of favipiravir docked with the Chandipura virus glycoprotein G (PDB ID: 5MDM).

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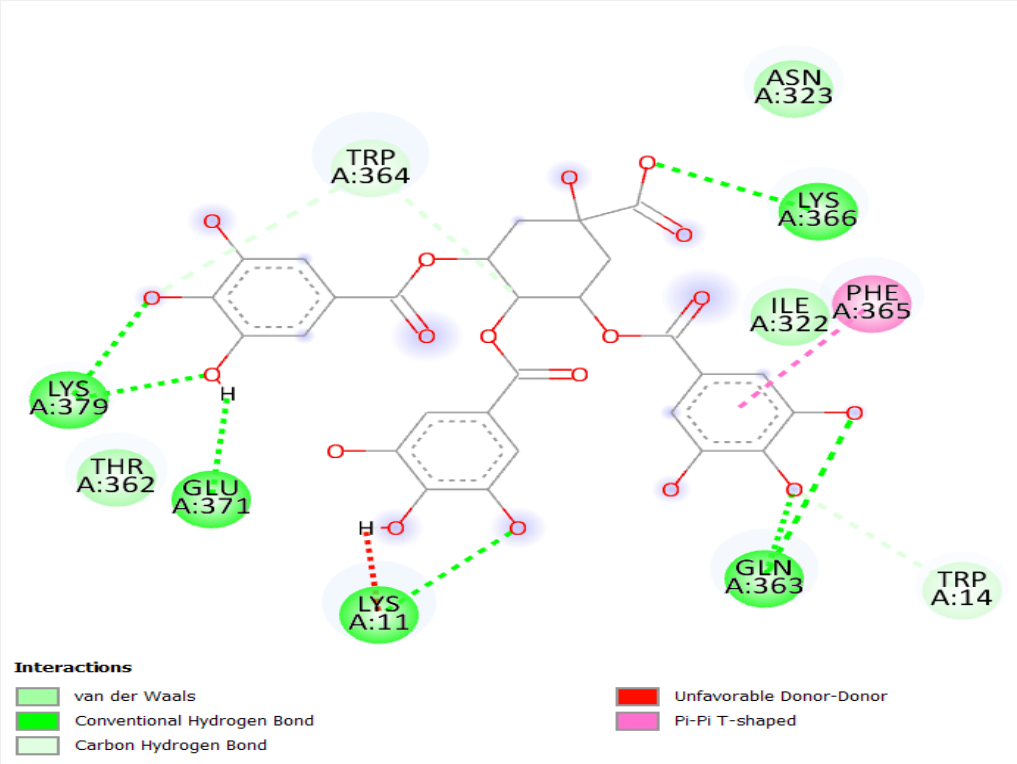

Figure 4: Two-dimensional interaction diagram of 3,4,5-tri-O-galloylquinic acid docked with the Chandipura virus glycoprotein G (PDB ID: 5MDM).

Favipiravir formed a stable interaction with the 5MDM glycoprotein G through four conventional hydrogen bonds involving Asn323 (two hydrogen bonds) and Gly368 (two hydrogen bonds). In addition, the fluorine atom of favipiravir established a halogen bond with Glu405, while a π-anion interaction was also observed with Glu405, contributing to ligand stabilization within the binding pocket. Furthermore, Val321, Gly324, Tyr367, and Lys366 participated in van der Waals interactions, supporting the overall stability of the protein–ligand complex Figure 3.

3,4,5-tri-O-galloylquinic acid exhibited the strongest interaction with the 5MDM glycoprotein G, forming an extensive hydrogen-bonding network with Lys379, Glu371, Lys11, Gln363, and Lys366. Additional carbon-hydrogen bonds were observed with Trp364 and Trp14, while a π–π T-shaped interaction was formed with Phe365, further stabilizing the ligand within the binding pocket. Asn323, Ile322, and Thr362 contributed through van der Waals interactions. One unfavorable donor–donor interaction was detected with Lys11, indicating a minor steric or electrostatic clash Figure 4.

Swiss ADME and boiled egg analysis

Swiss ADME analysis demonstrated distinct pharmacokinetic characteristics. 3,4,5-tri-O-galloylquinic acid exhibited a molecular weight of 648.48 g/mol, a TPSA of 318.50 $Å^2$, and a consensus Log P value of −0.37, indicating a highly polar compound with low lipophilicity. Pharmacokinetic prediction indicated low gastrointestinal absorption, no blood-brain barrier permeation, and that the compound is a P-glycoprotein substrate. Drug-likeness analysis revealed multiple violations of Lipinski, Ghose, Veber, Egan, and Muegge rules, resulting in a low predicted bioavailability score of 0.11. Additionally, one PAINS alert and two Brenk alerts were identified, while the synthetic accessibility score was 4.99.

In contrast, favipiravir displayed more favorable pharmacokinetic properties. The compound possessed a molecular weight of 157.10g/mol, TPSA of 88.84 $Å^2$, and a consensus Log P of −0.40. It was predicted to be highly water-soluble and exhibited high gastrointestinal absorption without blood-brain barrier permeation. Favipiravir was not predicted to be a P-glycoprotein substrate and fully complied with Lipinski's Rule of Five, with a bioavailability score of 0.55. No PAINS or Brenk alerts were detected, and the compound exhibited a low synthetic accessibility score (2.08), suggesting favorable medicinal chemistry characteristics. The Boiled-Egg model further confirmed these findings by predicting high intestinal absorption for favipiravir, whereas 3,4,5-tri-O-galloylquinic acid was predicted to have poor gastrointestinal absorption because of its high molecular weight and large polar surface area. Neither compound was predicted to permeate the blood-brain barrier Figure 5 & 6.

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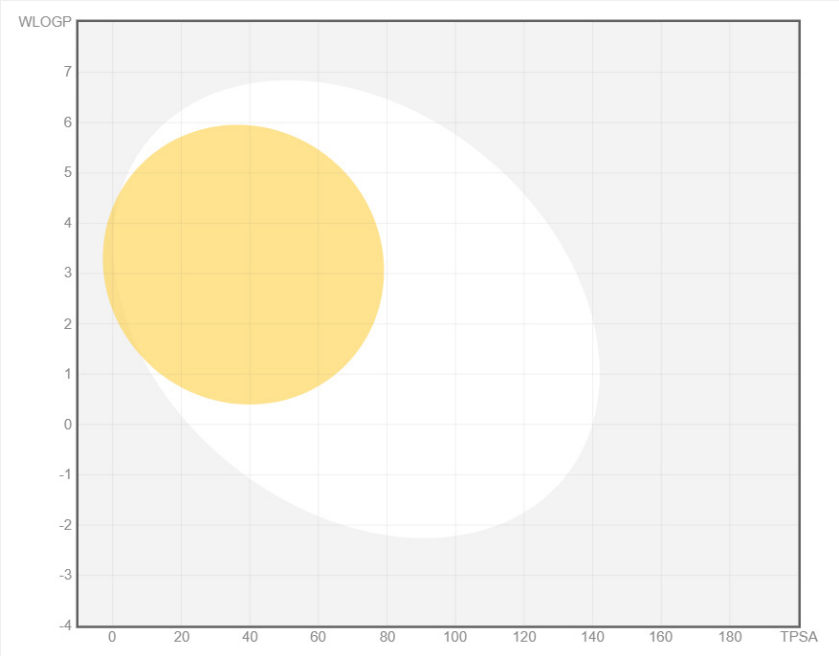

Figure 5: Boiled-Egg plot of 3,4,5-tri-O-galloylquinic acid.

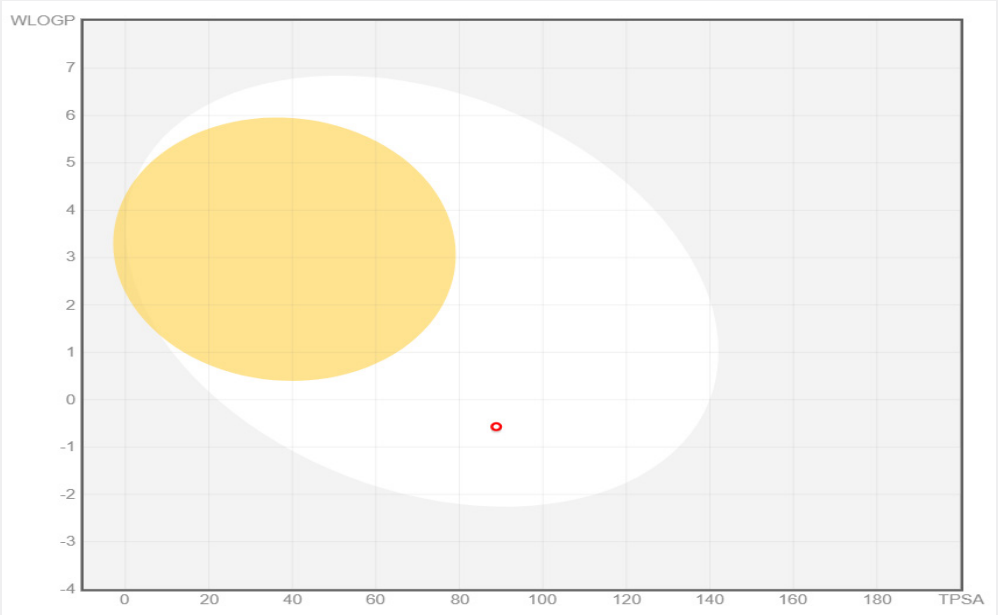

Figure 6: Boiled-Egg plot of favipiravir.

Despite the comparatively poor pharmacokinetic profile of 3,4,5-tri-O-galloylquinic acid, the compound demonstrated superior binding affinity toward Chandipura virus glycoprotein G in molecular docking studies compared with favipiravir. These findings suggest that while the phytochemical possesses promising target-binding characteristics, optimization through structural modification or advanced drug delivery systems may be necessary to improve its oral bioavailability.

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In silico toxicity prediction

The toxicity profile of 3,4,5-tri-O-galloylquinic acid was evaluated using the ProTox-3.0 server. The compound exhibited a predicted oral LD_{50} of 3700 mg/kg and was classified as Toxicity Class 5, indicating low acute oral toxicity. The compound was predicted to be inactive for hepatotoxicity, neurotoxicity, cardiotoxicity, carcinogenicity, immunotoxicity, mutagenicity, cytotoxicity, and ecotoxicity, suggesting a generally favorable safety profile. However, nephrotoxicity, respiratory toxicity, and clinical toxicity were predicted to be active with moderate probabilities, indicating that these endpoints should be considered in future experimental validation. Among the molecular initiating events, the compound showed predicted activity toward the thyroid hormone receptor α (THRα), transthyretin (TTR), and pregnane X receptor (PXR), whereas most other toxicity-associated molecular targets were predicted to be inactive. In cytochrome P450 metabolism analysis, activity was predicted only for CYP2C9, while CYP1A2, CYP2C19, CYP2D6, CYP3A4, and CYP2E1 were predicted to be inactive.

Discussion

Chandipura virus (CHPV), a member of the Rhabdoviridae family, is an emerging neurotropic virus responsible for outbreaks of acute encephalitis syndrome with high mortality, particularly among children. Despite its clinical significance, no specific antiviral therapy has been approved for the treatment of CHPV infection. Therefore, the identification of novel antiviral compounds targeting essential viral proteins remains an important strategy for therapeutic development [13]. The viral glycoprotein G is responsible for host cell attachment and membrane fusion, making it a promising target for antiviral drug discovery. In the present study, 3,4,5-tri-O-galloylquinic acid exhibited stronger binding affinities toward both glycoprotein G structures (4D6W and 5MDM) than the reference antiviral favipiravir. The docking scores of −4.7kcal/mol (4D6W) and −6.0kcal/mol (5MDM) suggest a greater binding affinity compared with favipiravir, which showed docking scores of −3.4kcal/mol and −4.0kcal/mol, respectively. Protein–ligand interaction analysis supported the docking results, showing that 3,4,5-tri-O-galloylquinic acid formed multiple hydrogen bonds with key active-site residues, including Lys86, Asn84, Asn163, Thr160, Ile161, His162, Lys366, Gln363, Lys379, and Glu371, along with π–π and π–alkyl interactions that stabilized the protein–ligand complex. In comparison, favipiravir formed fewer hydrogen bonds and hydrophobic interactions, which is consistent with its lower binding affinity.

Swiss ADME analysis revealed that favipiravir possesses superior drug-like properties, including high gastrointestinal absorption, compliance with Lipinski's Rule of Five, and better predicted oral bioavailability. In contrast, 3,4,5-tri-O-galloylquinic acid showed low gastrointestinal absorption, multiple drug-likeness rule violations, and poor predicted bioavailability owing to its high molecular weight and large polar surface area [14,15]. The Boiled-Egg model further predicted that the phytochemical is a P-glycoprotein substrate and is unlikely to cross the blood–brain barrier.

Although Swiss ADME and the Boiled-Egg model predicted that neither 3,4,5-tri-O-galloylquinic acid nor favipiravir is likely to cross the blood–brain barrier (BBB) through passive diffusion, this finding should be interpreted with caution. Chandipura virus is a highly neurotropic pathogen that causes acute encephalitis after invading the central nervous system, and BBB disruption during infection has been proposed as one of the mechanisms facilitating viral entry into the brain. Consequently, changes in BBB integrity during disease may also influence drug penetration into the brain. Furthermore, despite its predicted poor passive BBB permeability, favipiravir has demonstrated antiviral efficacy in experimental Chandipura virus-infected mouse models, significantly reducing viral loads in brain tissue and improving survival. Therefore, the inability to cross an intact BBB predicted by Swiss ADME should not be interpreted as definitive evidence of poor therapeutic efficacy [16,17]. Nevertheless, the limited predicted BBB permeability of 3,4,5-tri-O-galloylquinic acid may represent a challenge for treating the neurological manifestations of Chandipura virus infection and warrants further investigation through *in vivo* studies. ProTox-3.0 predicted that 3,4,5-tri-O-galloylquinic acid has low acute oral toxicity (LD_{50} = 3700 mg/kg, Toxicity Class 5) and is inactive for hepatotoxicity, carcinogenicity, mutagenicity, neurotoxicity, and cardiotoxicity. However, the predicted nephrotoxicity and respiratory toxicity warrant further experimental investigation before therapeutic application.

Conclusion

This study demonstrated that 3,4,5-tri-O-galloylquinic acid exhibited stronger binding affinity toward the Chandipura virus glycoprotein G than the reference antiviral favipiravir, supported by extensive hydrogen-bonding and hydrophobic interactions within the binding pocket. These findings suggest that the phytochemical has promising potential as a lead molecule for anti-Chandipura virus drug discovery. Swiss ADME and Boiled-Egg analyses indicated that, despite its superior docking performance, 3,4,5-tri-O-galloylquinic acid possesses poor predicted oral bioavailability, low gastrointestinal absorption, and limited passive blood–brain barrier permeability, whereas favipiravir demonstrated more favorable pharmacokinetic properties. In addition, ProTox-3.0 predicted a favorable safety profile for the phytochemical, characterized by low acute oral toxicity and no major predicted hepatotoxic, mutagenic, carcinogenic, or cardiotoxic effects. 3,4,5-tri-O-galloylquinic acid represents a promising natural lead compound against Chandipura virus glycoprotein G. Nevertheless, further optimization of its pharmacokinetic properties and validation through molecular dynamics simulations, cell-based antiviral assays, and *in vivo* studies are essential to establish its efficacy and therapeutic potential, particularly for the treatment of the neurological manifestations of Chandipura virus infection.

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