Global Journal of
Pharmacy & Pharmaceutical Sciences
ISSN: 2573-2250

Juniper Publishers
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Research Article
Volume 11 Issue 3 : February 2024
DOI: 10.19080/GJPPS.2024.11.555813

Glob J Pharmaceu Sci

Molecular Interactions Studies of Hepatitis C Drugs Inform Their Potential Use in Hepatitis B Treatment

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Submission: February 12, 2024; **Published:** February 20, 2024

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Abstract

The Hepatitis B virus is a DNA virus with relaxed circular strands. Among all hepatitis virus infections, the most severe and alarming is the Hepatitis B virus (HBV) infection, with approximately 1.5 million people getting infected each year, and approximately two people die every minute with HBV complications. Despite vaccination efforts, no complete cure exists, as viral DNA is integrated into the host genome and continuously produces the RNA and proteins that are further involved in viral assembly and infect healthy hepatocytes. Recent studies have highlighted the role of sodium taurocholate co-transporting peptide (NTCP) receptors in HBV infection. A study of molecular interactions of viral protein PreS1, which exclusively binds to the NTCP receptor, could lead to the development of compounds that inhibit the receptor, leading to the inhibition of viral internalization. The present study investigates molecular interactions between NTCP and various market-available HCV drugs using molecular docking techniques. Specifically, this study focuses on understanding the reason behind the failure of antivirals to provide a functional cure for HBV infections. Receptor interactions were studied using UCSF Chimera 1.17.3 version and Pymol. Autodock Vina was used for docking. The HCV nonstructural protein inhibitors and drugs that exhibited effective NTCP inhibition were used in docking studies. The docking studies' results demonstrated that specific HCV NS3/4A protease inhibitors could be potent NTCP receptor inhibitors. These interactions were characterized by favorable docking scores and low RMSD values, indicating strong molecular binding. Further research in this area holds promise in improving treatment outcomes and paving the way for a functional cure for Hepatitis B.

Keywords: Hepatitis B; Molecular docking; NTCP receptors; pre-S1 protein; UCSF chimera; Pymol; Docking Score

Abbreviations: HBV: Hepatitis B virus; DAAs: Direct-Acting Antivirals; rcDNA: relaxed circular DNA; cccDNA: covalently closed circular DNA

Introduction

Viral hepatitis poses a significant human health risk, leading to substantial morbidity and mortality. There are five hepatitis viruses - A, B, C, D, and E, each with unique genomic structures, replication methods, and clinical implications. Hepatitis A (HAV) and E (HEV) infections typically exhibit transient effects. Whereas hepatitis B (HBV), C (HCV), and delta (HDV) viruses can manifest as transient or chronic conditions. Furthermore, hepatocellular carcinoma, ranking among the top ten most prevalent cancers globally, shows a strong correlation with hepatitis B. In some geographic regions, there is an observable link between hepatocellular carcinoma and hepatitis C virus infections. Both HBV and HCV present a broad range of clinical conditions, spanning from asymptomatic carriers to the progression of liver cirrhosis, ultimately leading to the development of hepatocellular carcinoma [1-3]. The classification of hepatitis viruses and their genome is provided in Table 1. At present, there is no cure for HBV; it only can be managed, and a vaccine is available for the prevention of the infection. In contrast, HCV has no vaccine to prevent infection. However, the new treatments can potentially cure and prevent long-term complications.

HCV Pathogenesis and Treatment

The genome of HCV consists of a 9.6 kb single-stranded RNA of positive polarity. The virus exists as a free particle or is surrounded by host low-density lipoproteins, it attaches to the cell membrane of the target cells. The virus enters the target cells via clathrin-mediated endocytosis and releases the RNA genome, which gets translated to a polyprotein precursor at the rough endoplasmic reticulum. This precursor undergoes cleavage in

the presence of cellular and viral proteases to yield a set of ten matured proteins, including envelope glycoproteins and structural and non-structural proteins mediating a variety of virus-host interactions [4,5]. The various proteins and their functions are summarized in Table 2. The treatment of HCV infection involves direct-acting antivirals (DAAs), which target the nonstructural proteins mediating multiple steps in HCV replication. There are three classes of DAAs: NS3 protease inhibitors, NS5A inhibitors, and NS5B polymerase inhibitors. They are used individually or in combination therapy [6].

HBV Pathogenesis and Treatment

The genome of HBV comprises a relaxed circular DNA (rcDNA) with a complete minus strand and an incomplete plus strand. Host hepatocyte entry of HBV includes attaching the virus to the cell surface via binding to heparan sulfate proteoglycans in a low-affinity manner, which is followed by a high affinity binding to sodium taurocholate co transporting peptide (NTCP), a 343 amino acid peptide (Figure 1), which is exclusively expressed in the liver. This interaction triggers the internalization of the virus by endocytosis. The oligomerization status of NTCP modulates the virus internalization. The relaxed, close DNA is converted to covalently closed circular DNA (cccDNA) in the presence of several factors and acts as a template to transcribe four different lengths of RNAs. The host RNA polymerase II mediates this transcription. HBV enters the cell by endocytosis and gets released into the cytoplasm, then the nucleocapsid enters the nucleus. The 5' end of the minus strand of rcDNA consists of a pol-linked terminal redundant sequence and RNA oligonucleotide attached at the 5' end of the plus strand. The pol is linked covalently via a tyrosyl phosphodiester bond, and hence, tyrosyl DNA phosphodiesterase tends to play a role in the formation of cccDNA [7-9]. The cccDNA is formed by the removal of this pol-linked sequence and the RNA oligonucleotide from rcDNA followed by filling the gaps and ligation of the strands [10,11]. Currently, the treatment for chronic hepatitis B includes antiviral medications, which act by competing with the natural nucleotide (s)ides, interferon injections, and ultimately, the complications leading to liver transplant [12,13]. Recent studies have highlighted the significance of NTCP in the internalization of the virus. This newfound understanding has paved the way for research exploring NTCP as a viable target for developing novel treatments. Several FDA-approved compounds were potent inhibitors of NTCP-L-HBs interactions FDA [12, 14,15]. Developing compounds directly targeting cccDNA or inhibiting its formation could achieve a functional cure for hepatitis B [16]. The current treatment of Hepatitis B infection includes drug classes such as DAAs, protease inhibitors, and nucleotide analogs, which fail to provide a functional cure. These drug classes work on the RNA and the DNA products but not on the cccDNA and NTCP receptors. The study focuses on the molecular interactions between the NTCP domain and HBV pre-S1 protein containing 55 amino acid residues, which are crucial for viral internalization. Table 3 summarizes the envelope proteins targeted in treating HCV and HBV infections, and Table 4 summarizes the current treatments for HBV and HCV infections. The study's main objective is to evaluate the market-available HCV drugs using molecular docking techniques for their potential as NTCP receptors.

Materials and Methods

The molecular interactions between the NTCP receptor and the PreS1 region of HBV were studied using UCSF Chimera [17] and Pymol [18] software. The HCV drugs and FDA-approved compounds that exhibit inhibitory effects on NTCP receptors were chosen as ligands for docking studies. The 3D structures of protein and ligand molecules were obtained from the PDB and PubChem databases, respectively. The molecular docking of the drugs was carried out using Autodock Vina. The PDB and Pub Chem codes for the proteins and ligands are listed in Table 5. These structures were used for docking and studying the interactions. The RMSD (Root mean square deviation) is commonly employed to assess the accuracy of replicating a known binding pose. A low RMSD relative to the actual binding pose is favorable, ideally below 1 Angstrom (A°).

$$RMSD = \sqrt{\frac{\sum_i d_i^2}{n}}$$

The RMSD value, where "d" represents the distance between each of the "n" pairs of equivalent atoms in two optimally superposed structures, is zero for identical structures. As the structures diverge, the RMSD values increase [19].

Results

The analysis of the molecular interactions at the binding interphase between the NTCP receptor and pre-S1 HBV protein identified key amino acids essential for binding. The binding of the pre-S1 viral protein with the NTCP receptor involves fourteen amino acids, which bind with high affinity (bond length ≤ 3.5 A°). The NTCP receptor amino acids that bind to the viral protein with high affinity are aspartic acid 152, lysine-153, glutamine-264, glycine-19, lysine-20, valine 272, alanine-273, asparagine-87 and isoleucine-88. Table 6 summarizes the list of amino acids involved in binding with their bond lengths, and Figures 2 & 3 depicts the molecular interactions between the NTCP receptor and pre-S1 protein. Molecular docking studies revealed that the HCV NS3/4A protease inhibitors exhibited a higher docking score with NTCP receptors compared to HCV NS3/4A protease. The compounds irbesartan, zafirlukast, ezetimibe, and vanitaricin A have shown a favorable docking score with NTCP receptors compared to HCV NS3/4A protease. The HCV NS5A and NS5B polymerase inhibitors did not exhibit a favorable docking score with NTCP receptors. Antiviral drugs such as tenofovir, adefovir, entecavir, and lamivudine have exhibited a lower docking score with NTCP receptors. Table 7 summarizes the docking scores for drugs docked with HCV nonstructural proteins and NTCP receptors. The molecular interactions between various drugs and the receptors are depicted in Figures 4-7.

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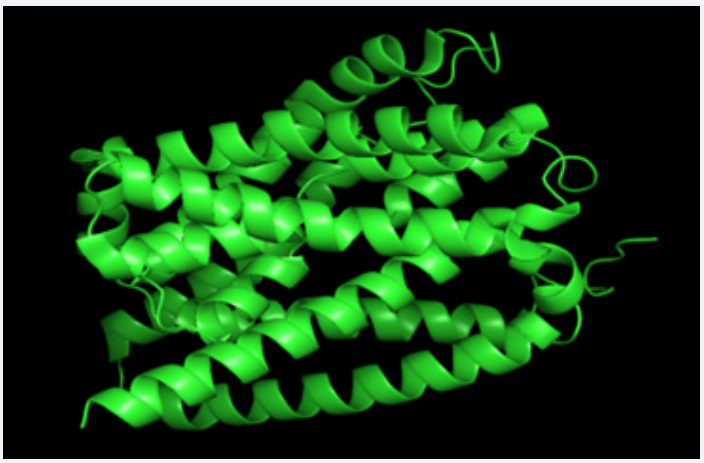

Figure 1: Structure of Sodium taurocholate co transporting polypeptide.

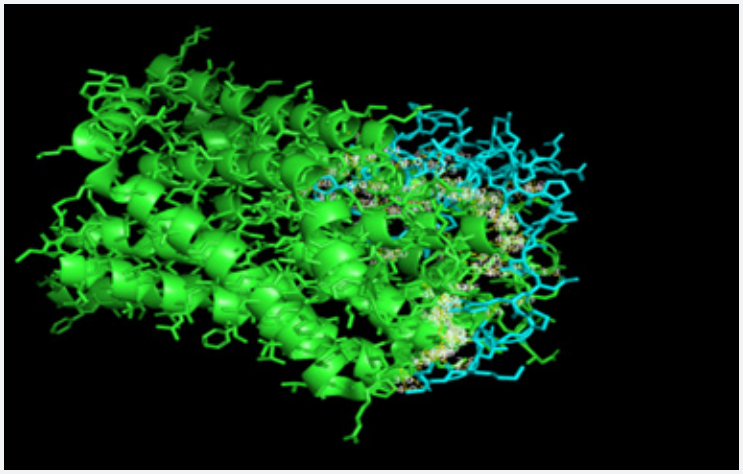

Figure 2: HBV PreS1 protein (Blue) binding to NTCP receptor (Green) with several interactions with bond lengths ≤ 4.0 A°.

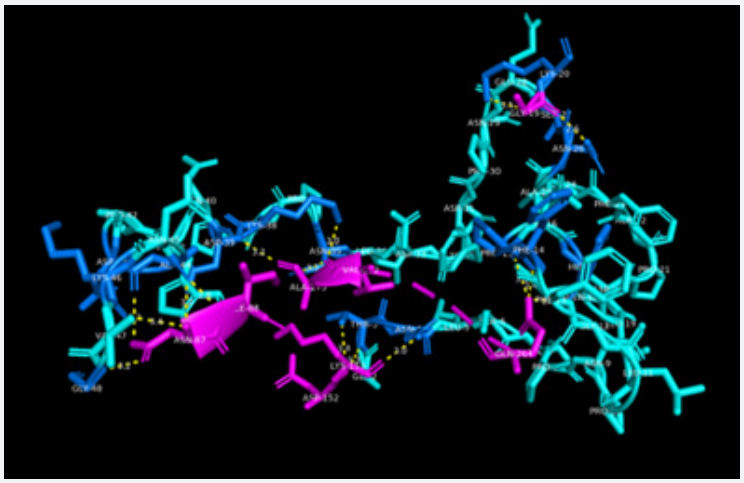

Figure 3: Polar interactions between the amino acids (Dark blue) of the HBV PreS1 region and NTCP amino acids (Magenta) with bond lengths ≤ 3.5 A°.

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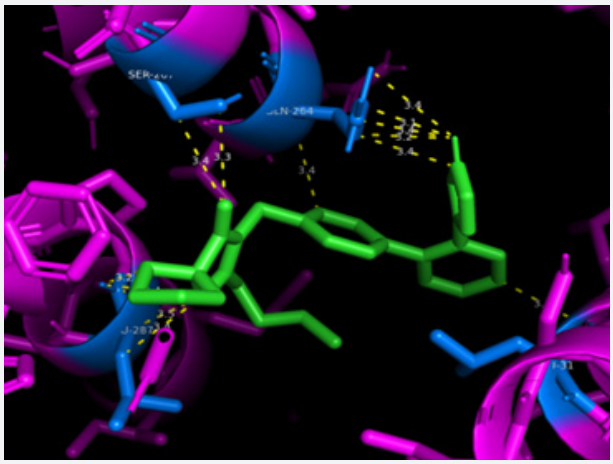

Figure 4: Irbesartan interaction with NTCP.

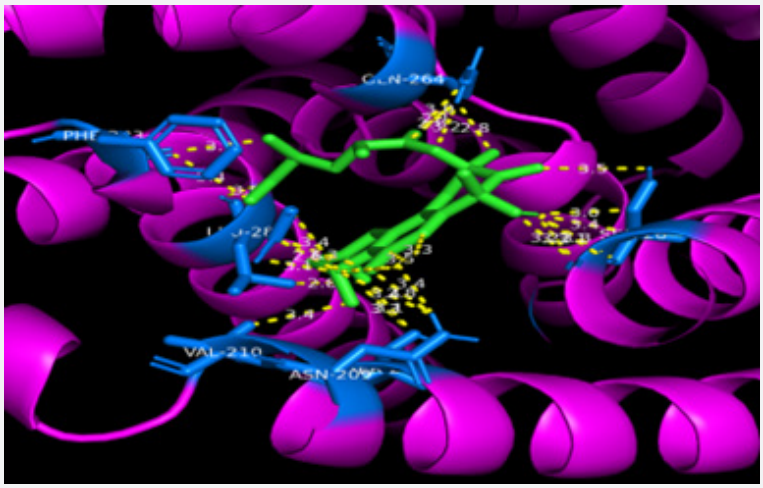

Figure 5: Vanitaracin A interactions with NTCP.

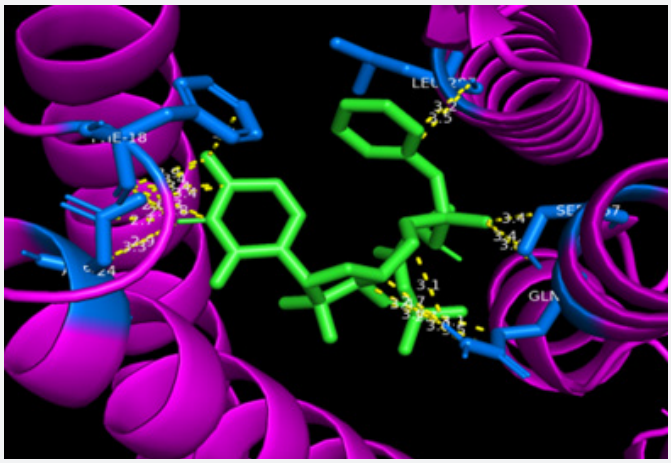

Figure 6: Sofosbuvir HTCP interactions.

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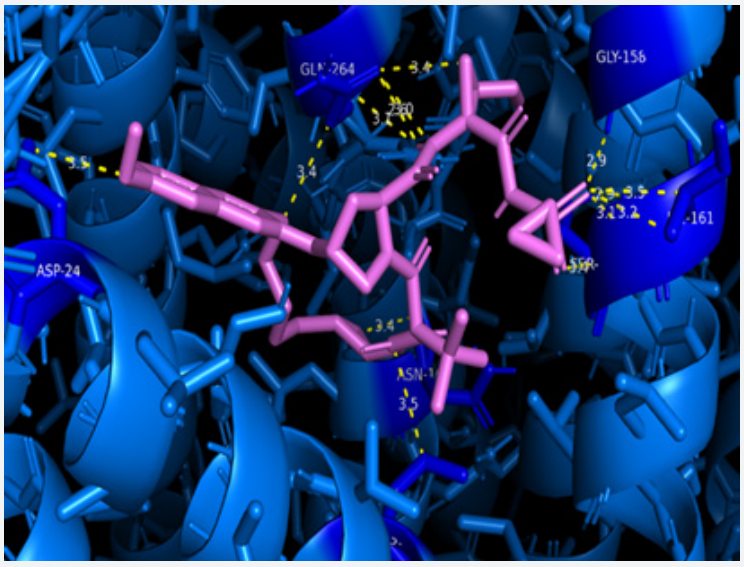

Figure 7: Grazoprevir interactions with NTCP.

Table 1: Classification of Hepatitis Viruses.

Virus	Genome	Transmission	Prevention	Treatment
Hepatitis A	Unenveloped single positive-stranded RNA of 7478 bases	Eating contaminated food or drinking contaminated water	Vaccination Practicing good hygiene	No treatment
Hepatitis B	Enveloped double-stranded DNA of 3226 base pairs	Through contact with the blood or bodily fluids of an infected person	Vaccination Practicing good hygiene Blood Screening	Alpha interferon peginterferon
Hepatitis C	Enveloped single positive-stranded RNA of at least 10,000 bases	Blood-to-blood contact	Practicing good hygiene Avoid sharing needles, toothbrushes, razors, or nail scissors	Direct-acting antiviral drugs
Hepatitis D	Single-stranded circular RNA of 1167 nucleotides	Contact with infected blood. (only occurs in people already infected with hepatitis B)	Hepatitis B Vaccination. Avoid sharing needles, toothbrushes, razors or nail scissors	Interferon
Hepatitis E	Non-enveloped single – stranded RNA	Eating contaminated food or drinking contaminated water	Practicing good hygiene Avoid drinking water that has come from a potentially unsafe source	No treatment

Table 2: HCV proteins.

HCV Proteins	Functions
Structural Proteins	
HCV Core	RNA binding, immune modulation, cell signaling, oncogenic potential, autophagy
HCV E1/E2 envelope glycoproteins	Viral attachment, entry, and fusion. Renders the antibody responses ineffective and contribute to virus persistence.
Nonstructural (NS) proteins	
NS2	Viral assembly
NS3 protease	HCV processing by cleaving NS3 into 4 proteins
NS4B	Mediates virus-host interactions
NS5A and NS5B	HCV RNA replication and translation

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Table 3: HBV Proteins.

HBV Proteins	Functions
Envelope protein pre-S1	Viral internalization
Envelope protein pre-S2	Unknown
Envelope protein S	Unknown

Table 4: Current drugs in the treatment of HBV and HCV infections.

HCV Drugs		
Direct Acting Antivirals (DAAs)	NS3/4 protease inhibitors	Boceprevir, Telaprevir, Paritaprevir, Simeprevir, Asunaprevir, Grazoprevir
	NS5A inhibitors	Daclatasvir, Ledpasvir, Ombitasvir, Elbasvir, velpatasvir, Ritonavir
	NS5B polymerase inhibitors	Sofosbuvir, Dasabuvir
Immunomodulators	Interferon	Peginterferon alpha2a
HBV Drugs		
Antivirals	Nucleot(s)ide Analogues	Tenofovir, Entecavir, Adefovir, Lamivudine
Immunomodulators	Interferons	Pegylated Interferon, Interferon Alpha

Table 5: PDB and Pub Chem Codes for the Protein and Ligand Structures.

Protein Structure	PDB Code
NTCP receptor protein with PreS1 peptide	8HXR
Human NTCP receptor protein	7VAD
HCV NS3/4A protease	3KF2
HCV NS5B polymerase	6MVQ
Drug compounds	**Pub Chem ID**
Boceprevir	10324367
Sofosbuvir	45375808
Dasabuvir	56640146
Grazoprevir	44603531
Lamivudine	60825
Paritaprevir	45110509
Ezetimibe	150311
adefovir	60172
tenofovir	464205
entecavir	135398508
Zafirlukast	5717
Irbesartan	3749
Vanitaracin A	122190346

Table 6: The polar interactions between the HBV Pre S1 protein and the human NTCP receptor.

HBV Pre S1 Peptide Chain		NTCP Receptor Protein		
Amino acid	**Position**	**Amino acid**	**Position**	**Bond length(A°)**
Threonine	3	Aspartic Acid	152	3.3
Threonine	3	Aspartic Acid	152	3.4
Asparagine	4	Lysine	153	3

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Phenylalanine	14	Glutamine	264	2.4
Proline	15	Glutamine	264	3.3
Histidine	17	Glutamine	264	2.9
Asparagine	26	glycine	19	2.6
Serine	27	Lysine	20	3.5
Asparagine	35	Valine	272	3.1
Lysine	38	Valine	272	3
Aspartic Acid	39	Alanine	273	3.2
Aspartic acid	43	Asparagine	87	3.2
Alanine	44	Asparagine	87	3.2
Lysine	46	Asparagine	87	3
Glycine	48	Asparagine	87	3.2

Table 7: Docking scores of HCV drugs with NTCP receptor.

Drug Category	Docking Scores with HCV Nonstructural proteins	Docking Scores with NTCP receptor
NS3/4A protease inhibitors		
Boceprevir	-7	-7.6
Grazoprevir	-7.4	-9.1
Paritaprevir	-7	-8
NS5A inhibitors		
Ritonavir	-8.5	-8
NS5B polymerase inhibitors		
Sofosbuvir	-8	-6.9
Dasabuvir	-9.7	-8.6
Nucleot(s)ide Analogues		
Tenofovir	-6.5	-6.1
Adefovir	-5.7	-5.7
Entecavir	-7.9	-6.2
Lamivudine	-6	-5.2
NTCP Receptor inhibitors		
Zafirlukast	-8.2	-9.4
Irbesartan	-6.8	-8.3
Ezetimibe	-7.1	-7.7
Vanitaracin A	-5.8	-7.8

Discussion

The observations of the molecular interactions between NTCP and pre-S1 revealed that the amino acids asparagine-87, aspartic acid-152, lysine 153, glutamine-264, glycine-19, valine-272, and alanine-273 of the NTCP receptors play a vital role in the viral protein binding. The drugs that are used in the treatment of HBV, such as tenofovir, adefovir, entecavir, and lamivudine, which are nucleotide analogs, did not bind to the NTCP receptor with good binding affinity. these compounds did not exhibit any significant interactions with NTCP amino acids. The NS3/4A protease inhibitors such as boceprevir, grazoprevir, and paritaprevir have exhibited favorable docking scores and strong interactions with glutamine-264, aspartic acid-153, asparagine-87 emphasizing these amino acids' significance in NTCP receptor inhibition. Additionally, these drugs, when docked with NS3/4A protease, exhibited interactions with the amino acid glutamine-41 in these receptors. These findings suggest that the NS3/4A protease inhibitors, particularly those with substantial interactions with amino acid glutamine hold promise as potential NTCP receptor inhibitors. Zafirlukast, a leukotriene receptor antagonist has exhibited a very favorable docking score of -9.4 and multiple interactions with amino acid arginine. Several studies revealed its potential as an NTCP receptor inhibitor [20]. Drugs such as ezetimibe, an antihyperlipidemic agent, and irbesartan (Figure 6), an angiotensin II receptor agonist, exhibited good docking scores and exhibited interaction with amino acids that were significantly involved in the receptor binding. Several research studies

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have shown that these compounds are efficient NTCP receptor inhibitors [21-23]. Vanataricin A, a polyketide, exhibits a good docking score and interacts with nine amino acids of the receptor, including multiple interactions with glutamine-264. Studies also demonstrated that this drug explicitly inhibits hepatitis B virus entry by targeting NTCP receptors [24]. All the compounds with a docking score of -7.0 and above exhibited multiple interactions with the amino acid glutamine-264, asparagine-87, and asparagine-262, making this interaction significant for receptor inhibition.

Conclusion

The molecular docking studies showed the constraints of currently available antiviral therapies in effectively combating HBV infection. The market-available drug molecules that exhibit binding patterns akin to the pre-S1 viral protein on NTCP receptors demonstrated favorable docking scores, pointing to their potential as potent NTCP receptor inhibitors. These findings suggest a promising avenue for developing more effective treatment for hepatitis B.

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