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Sequence Analysis, DNA Methylation, and Molecular Therapy of Oral Cancer Caused by Human Papilloma Virus

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Abstract

DNA methylations with oral cancer are being associated with high-risk Human Papillomavirus (HPV) and Epstein–Barr Virus (EBV) onco-proteins interactions may cooperate to increase disease severity. The work focused on human papillomavirus strain 16, a high-risk, sexually transmitted responsible for 95% of all cervical cancers and a large portion of oropharyngeal cancers. The prediction of genes from has shown seven genes from HPV16 strain. Based on gene identification studies, Human papilloma virus type 16 has shown proteins like E6 protein, E7 protein, E1, E2, E5, L1, and L2 proteins. Methylation frequency has shown for APC, BRCA1, CDH1, DAPK1 and RARB that are linked to oral cancer and also a key tumor suppressor gene via promoter hypermethylation that is robustly accelerated by HPV16 oncoproteins. Based on methylation frequency, CDKN2A, MGMT, CDH1, CDKN2B genes for oral cancer are related to squamous cell carcinoma. Protein alignment of E6, E7 (HPV16 strain) and CDH1, by SPDBV v3.7 has shown structural similarity. Highest methylation frequency was shown with CDH1 based on molecular therapy for oral cancer. Human Papillomavirus 16 (HPV16) is highly pathogenic because of its early proteins, E6 and E7 that act as potent oncoproteins. Hence the target proteins E6 and E7 are being selected against drug molecules in the present study. The drug compounds – like Cisplatin, Carboplatin, 5-fluorouracil, Paclitaxel, Docetaxel, and Hydroxyurea – have been analyzed for control of HPV16 and oral cancer based on in silico analysis. The present study has shown that Paclitaxel and Docetaxel are better molecular compounds for the control of HPV16 and oral cancers based on in silico analysis. Further in vivo and in vitro analysis may provide better mechanism and treatment methods for oral cancer.

Keywords: DNA methylation, HPV16 strain, oral cancer, paclitaxel, docetaxel

INTRODUCTION

Oral cancer (also known as Oropharyngeal cancer) is a disease formed in the tissues of the mouth affecting humans as malignant (cancer) disease [1]. Human papillomavirus (HPV) can increase the risk of oral cancer due to betel quid chewing, cigarette smoking and alcohol [2]. Infection by HPV type 16 or 18 are being greatly increased the risk of cancers worldwide [3–5].

Oral cancer is a type of head and neck cancer, occur in oropharyngeal cancers (cancers of the back of the throat, tonsils, and base of the tongue) at the time of HPV infection [6–8]. Most oropharyngeal cancers (more than 90%) are generally squamous cell carcinomas that have cell lining of the oropharynx. Cetuximab, Cisplatin, Pembrolizumab, nivolumab, Carboplatin, 5-fluorouracil, Docetaxel (Taxotere), Paclitaxel (Taxol), Hydroxyureaare some of the compounds using in the treatment of oral cancer [9].

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A common cancer type associated with viruses with E6 and E7 protein makes dysregulated expression of the CDH1 gene, that encodes E-cadherin and activates DNA methyltransferases (DNMTs) [10–13]. Before the development of malignant disease, DNA methylation in about 68 HIV-seropositive in *APC*, *CDH1*, *CDH13*, *CDKN2A*, *CDKN2B*, *DAPK1*, *FHIT*, *GSTP1*, *HIC1*, *MGMT*, *MLH1*, *RARB*, *RASSF1*, *TERT* and *TIMP3*are the transcription start site that may generally lead to increase in $p16^{INK4A}$/$p14^{ARF}$ expression. DNA methylation with malignancies due to Human Papilloma Virus Linked with Epstein–Barr Virus and several viruses was reported in previous research [4, 5]. The 10 genes – like *ATP2A1*, *CALML5*, *GNMT*, *MAL*, *DNAJC5G*, *GPT*, *LYNX1*, *LY6D*, *MGC16275*, and *MRGPRF* – showed significant increase in recurrence of methylation groups causing oral cancer.

MATERIALS AND METHODS

The genomes of living organisms are complex with huge datasets and analyzing the big data is a novel task in the present analysis. The following servers and software's are applied in the present research work:

PAVE

PAVE is a database containing the data of HPV.

The Alphapapillomavirus 9, a Human Papillomavirus 16 (HPV16) with complete genome gi|333031|lcl|HPV16REF is retrieved from PAVE database (https://pave.niaid.nih.gov/#search/search_database/locus_view/fetch?id=HPV16REF&format=Locus%20view&hasStructure=none)

FGENESV0

FGENESV0 is the server that generally predicts genes from the viral genome.

BLASTp

BLASTp is a protein alignment server use for the identification of genes.

System properties

Intel ® core ™ i5 CPU processor with 650 @3.20GHz 3.20GHz; 8GB RAM, and 64-bit processor is used for the analysis of genes and other *in silico* processes.

Pubmeth

Pubmeth is a cancer methylation database that includes reporting of genes that are present in genomes of methylated cells in several cancer types like oral cancer.

dbGENVOC

dbGENVOC, a comprehensive, flexible database framework, developed with an aim to allow potential users to access, query, browse and download clinically relevant somatic and germline variation data from Indian oral cancer patients (http://research.nibmg.ac.in/dbcares/dbgenvoc/).

String v11.0

Protein–protein interactions that are predicted based on physical and functional associations can be done by using STRING v11.0 database. The search sites for multiple query protein interactions used are CDKN2A, MGMT, CDH1, and CDKN2B.

Retrieval of Ligands and Proteins

The drug molecules are retrieved from Drugbank (Table 1).

iGEMDOCK v2.1

iGEMDOCK v2.1 is an offline tool for virtual screening for ligands with selected proteins.

Table 1. Drugs for oral cancer from drug bank.

S. N.	Name	ChEBI
1	Cisplatin	DB00515.
2	Carboplatin	DB00958.
3	5-fluorouracil	DB00544.
4	Paclitaxel	Paclitaxel.
5	Docetaxel	DB01248.
6	Hydroxyurea	DB01005.

The mutated protein of tumor gene CDH1 is retrieved from string via PDB database (https://www.rcsb.org/)

RESULTS AND DISCUSSION

The prediction of genes using complete genome from FGENESV0 (http://www.softberry.com/berry.phtml?topic=index&group=programs&subgroup=gfind) has shown 7 genes from HPV16 strain (Table 2).

FGENESV0: Prediction of potential genes in viral genomes:

- *Time:* Tue Jan 1 00:00:00 2005.
- *Seq Name:* Test sequence.
- *Length of Sequence:* 7906 bp.
- *Number of Predicted Genes:* 7.

Table 2. Gene prediction of HPV16.

N S Start End Score
• 1 + CDS 83 – 559 477 • 2 + CDS 562 – 858 297 • 3 + CDS 865 – 2814 1950 • 4 + CDS 2756 – 3853 1098 • 5 + CDS 3850 – 4101 252 • 6 + CDS 4237 – 5658 1422 • 7 + CDS 5561 – 7156 1596

Predicted Protein(s)

>GENE 1 83 – 559 158 aa, chain +

MHQKRTAMFQDPQERPRKLPQLCTELQTTIHDIILECVYCKQQLLRREVYDFAFRDLCIVYRDG NPYAVCDKCLKFYSKISEYRHYCYSLYGTTLEQQYNKPLCDLLIRCINCQKPLCPEEKQRHLDKKQ RFHNIRGRWTGRCMSCCRSSRTRRETQL

>GENE 2 562 – 858 98 aa, chain +

MHGDTPTLHEYMLDLQPETTDLYCYEQLNDSSEEEDEIDGPAGQAEPDRAHYNIVTFCCK CDSTLRLCVQSTHVDIRTLEDLLMGTLGIVCPICSQKP

>GENE 3 865 – 2814 649 aa, chain +

MADPAGTNGEEGTGCNGWFYVEAVVEKKTGDAISDDENENDSDTGEDLVDFIVNDNDYL TQAETETAHALFTAQEAKQHRDAVQVLKRKYLGSPLSDISGCVDNNISPRLKAICIEKQSRAA KRRLFESEDSGYGNTEVETQQMLQVEGRHETETPCSQYSGGSGGGCSQYSSGSGGEGVSERH TICQTPLTNILNVLKTSNAKAAMLAKFKELYGVSFSELVRPFKSNKSTCCDWCIAAFGLTPSIA DSIKTLLQQYCLYLHIQSLACSWGMVVLLLVRYKCGKNRETIEKLLSKLLCVSPMCMMIEPP KLRSTAAALYWYKTGISNISEVYGDTPEWIQRQTVLQHSFNDCTFELSQMVQWAYDNDIVD DSEIAYKYAQLADTNSNASAFLKSNSQAKIVKDCATMCRHYKRAEKKQMSMSQWIKYRCD RVDDGGDWKQIVMFLRYQGVEFMSFLTALKRFLQGIPKKNCILLYGAANTGKSLFGMSLMK

FLQGSVICFVNSKSHFWLQPLADAKIGMLDDATVPCWNYIDDNLRNALDGNLVSMDVKHRP
LVQLKCPPLLITSNINAGTDSRWPYLHNRLVVFTFPNEFPFDENGNPVYELNDKWKSFFSRTW
SRLSLHEDEDKENDGDSLPTFKCVSGQNTNTL

>GENE 4 2756 – 3853 365 aa, chain +
METLCQRLNVCQDKILTHYENDSTDLRDHIDYWKHMRLECAIYYKAREMGFKHINHQVV
PTLAVSKNKALQAIELQLTLETIYNSQYSNEKWTLQDVSLEVYLTAPTGCIKKHGYTVEVQF
DGDICNTMHYTNWTHIYICEEASVTVVEGQVDYYGLYYVHEGIRTYFVQFKDDAEKYSKNK
VWEVHAGGQVILCPTSVFSSNEVSSPEIIRQHLANHPAATHTKAVALGTEETQTTIQRPRSEPD
TGNPCHTTKLLHRDSVDSAPILTAFNSSHKGRINCNSNTTPIVHLKGDANTLKCLRYRFKKHC
TLYTAVSSTWHWTGHNVKHKSAIVTLTYDSEWQRDQFLSQVKIPKTITVSTGFMSI

>GENE 5 3850 – 4101 83 aa, chain +
MTNLDTASTTLLACFLLCFCVLLCVCLLIRPLLLSVSTYTSLIILVLLLWITAASAFRCFIVYII
FVYIPLFLIHTHARFLIT

>GENE 6 4237 – 5658 473 aa, chain +
MRHKRSAKRTKRASATQLYKTCKQAGTCPPDIIPKVEGKTIADQILQYGSMGVFFGGLGIG
TGSGTGGRTGYIPLGTRPPTATDTLAPVRPPLTVDPVGPSDPSIVSLVEETSFIDAGAPTSVPSIP
PDVSGFSITTSTDTTPAILDINNTVTTVTTHNNPTFTDPSVLQPPTPAETGGHFTLSSSTISTHNY
EEIPMDTFIVSTNPNTVTSSTPIPGSRPVARLGLYSRTTQQVKVVDPAFVTTPTKLITYDNPAYE
GIDVDNTLYFSSNDNSINIAPDPDFLDIVALHRPALTSRRTGIRYSRIGNKQTLRTRSGKSIGAK
VHYYYDLSTIDPAEEIELQTITPSTYTTTSHAASPTSINNGLYDIYADDFITDTSTTPVPSVPSTS
LSGYIPANTTIPFGGAYNIPLVSGPDIPINITDQAPSLIPIVPGSPQYTIIADAGDFYLHPSYYMLR
KRRKRLPYFFSDVSLAA

>GENE 7 5561 – 7156 531 aa, chain +
MQVTFIYILVITCYENDVNVYHIFFQMSLWLPSEATVYLPPVPVSKVVSTDEYVARTNIYY
HAGTSRLLAVGHPYFPIKKPNNNKILVPKVSGLQYRVFRIHLPDPNKFGFPDTSFYNPDTQRL
VWACVGVEVGRGQPLGVGISGHPLLNKLDDTENASAYAANAGVDNRECISMDYKQTQLCLI
GCKPPIGEHWGKGSPCTNVAVNPGDCPPLELINTVIQDGDMVDTGFGAMDFTTLQANKSEVP
LDICTSICKYPDYIKMVSEPYGDSLFFYLRREQMFVRHLFNRAGTVGENVPDDLYIKGSGSTA
NLASSNYFPTPSGSMVTSDAQIFNKPYWLQRAQGHNNGICWGNQLFVTVVDTTRSTNMSLC
AAISTSETTYKNTNFKEYLRHGEEYDLQFIFQLCKITLTADVMTYIHSMNSTILEDWNFGLQPP
PGGTLEDTYRFVTSQAIACQKHTPPAPKEDPLKKYTFWEVNLKEKFSADLDQFPLGRKFLLQ
AGLKAKPKFTLGKRKATPTTSSTSTTAKRKKRKL (Table 3).

Table 3 shows identification of genes from Human papilloma virus type 16.

Table 3. Identification of genes by BLASTp.

Gene number	Gene name based on BLASTp
1	E6 protein [Human papillomavirus type 16].
2	E7 protein [Human papillomavirus type 16].
3	E1 [Human papillomavirus type 16].
4	E2 [Human papillomavirus type 16].
5	E5 protein [Human papillomavirus type 16].
6	L2 [Human papillomavirus type 16].
7	L1 [Human papillomavirus type 16].

Table 3 shows molecular diagnosis of HPV genes. The drugs Paclitaxel and Docetaxel show better activity against HPV16 strain (Tables 4–8) (Figures 1–4).

Table 4. Drug screening for HPV16.

Compound	E6		E7	
	Total energy in Kcal/mol	Active site	Total energy in Kcal/mol	Active site
Cisplatin	–47.08	H-M-GLU-48 H-M-VAL-69	–45.72	E-S-GLU-80 H-S-TYR-52 H-M-ASN-53 H-M-VAL-55 H-S-GLU-80 V-M-VAL-55
Carboplatin	–58.99	H-M-LEU-107 H-S-ARG-109 H-M-TRP-139 V-S-LEU-107 V-S-ARG-109 V-M-ARG-138 V-S-ARG-138	–67.08	H-M-CYS-59 H-S-CYS-59 H-M-LEU-83 H-M-MET-84 H-M-GLY-85 H-M-GLN-70 V-M-LEU-87 V-M-GLY-88
5-fluorouracil	–60.77	H-M-MET-8 H-M-GLN-10 H-S-GLN-10 H-M-ASP-11 H-M-GLU-14 H-S-GLU-14 H-S-GLU-25 V-S-ASP-11	–57.97	H-M-CYS-68 H-M-VAL-90 H-S-SER-95 V-M-LEU-67 V-S-LEU-67 V-M-CYS-91 V-M-PRO-92 V-S-PRO-92
Paclitaxel	*–114.75*	H-S-SER-150 H-M-ARG-151 H-M-ARG-154 H-M-GLN-157 H-S-GLN-157 V-M-ARG-151 V-M-ARG-153 V-M-ARG-154 V-M-GLU-155 V-S-GLU-155 V-M-GLN-157 V-S-GLN-157	*–102.86*	H-S-TYR-52 H-M-ASN-53 H-S-ASN-53 V-M-HIS-51 V-S-HIS-51 V-M-TYR-52 V-S-TYR-52V-M-ASN-53 V-S-ASN-53 V-S-LYS-97
Docetaxel	*–117.18*	H-S-ASP-56 H-M-ASP-105 H-M-LEU-107 H-M-PRO-116 H-M-LEU-117 H-M-CYS-118 H-S-CYS-118 V-S-LYS-18 V-M-PHE-54 V-S-PHE-54 V-S-ASP-56 V-S-LEU-107 V-M-PRO-116 V-S-PRO-116	*–98.78*	H-M-HIS-51 H-S-HIS-51 H-S-GLN-96 V-M-HIS-51 V-S-HIS-51 V-M-TYR-52 V-S-TYR-52 V-M-ASN-53 V-S-ARG-66 V-M-GLN-96 V-S-GLN-96
Hydroxyurea	–56.67	H-M-GLU-48 H-S-ASP-51 H-S-ARG-55 H-M-VAL-69 H-M-ASP-71 H-S-ASP-71	–44.39	H-M-GLN-70 H-S-CYS-59 H-M-GLY-85 H-M-THR-86 H-M-LEU-87 H-M-GLY-88

Figure 1 shows the docking sites of drugs with E6 and E7.

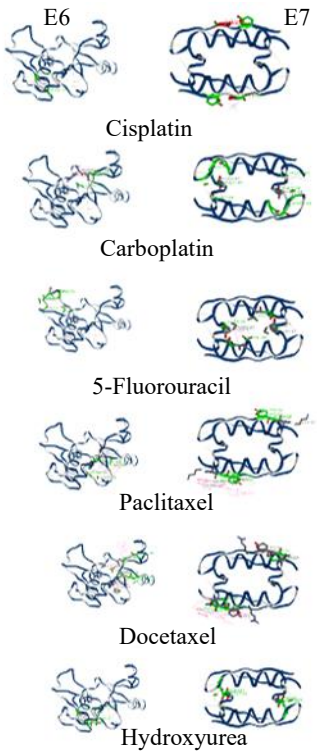

Figure 1. Drugs binding E6 and E7 of HPV16.

Table 5 shows that CDKN2A, CDH1, and CDKN2B are showing characteristic relationships with oral cancer.

Table 5. Genes are related to oral cancer with methylation and its summary from Pubmeth and dbGENVOC.

Methylated gene	Name as on pUBMETH	dbGENVOCData project	Variation
CDKN2A	Cyclin-dependent kinase inhibitor 2A, isoform 4 (p14ARF) (p19ARF)	*Gingivo-buccal oral squamous cell carcinoma (BM-TCGA: Buccal Mucosa; OC-TCGA: Oral Cavity; OT-TCGA: Oral Tongue)*	*Non sence:6; SNP:6*
RASSF1	Ras association domain-containing protein 1	*Head and Neck Squamous Cell Carcinoma (BM-TCGA: Buccal Mucosa; OC-TCGA: Oral Cavity; OT-TCGA: Oral Tongue)*	*Missence 1; SNP 1*
MGMT	Methylated-DNA-protein-cysteine methyltransferase (EC 2.1.1.63) (6-O-methylguanine-DNA methyltransferase) (MGMT) (O-6-methylguanine-DNA- alkyltransferase)	*Head and Neck Squamous Cell Carcinoma (OC-TCGA: Oral Cavity; OT-TCGA: Oral Tongue)*	*Missence 2: SNP:2*
CDH1	Epithelial-cadherin precursor (E-cadherin) (Uvomorulin) (Cadherin-1) (CAM 120/80) (CD324 antigen	*Gingivo-buccal oral squamous cell carcinoma*	Missence 1: SPN 2
CDKN2B	Cyclin-dependent kinase 4 inhibitor B (p14-INK4b) (p15-INK4b) (p15INK4B) (Multiple tumor suppressor 2) (MTS2)	*OC-TCGA: Oral Cavity*	Missence 1; SPN 1

Figure 2 shows protein alignment of CDH1, E6, and E7 by v3.7 SPDBV shown structural similarity.

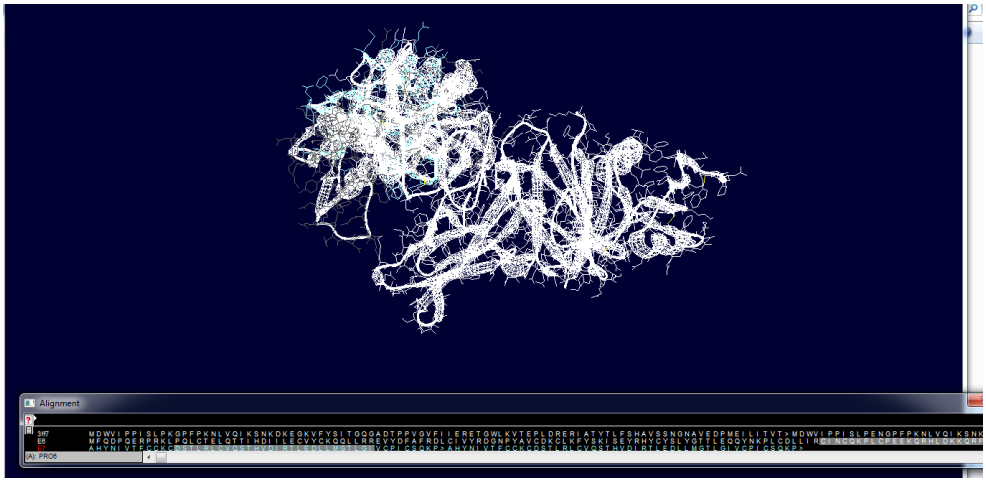

Figure 2. Protein alignment of CDH1, E6 and E7 by SPDBV v3.7.

Table 6. Methylated frequency and details in oral cancer genes using Pubmeth.

Gene	Number of references	Number of references in gastric cancer	Number of samples	Methylation frequency	Details for methylation In Gastric
CDKN2B	205	4	161	21	No subtype specified (2), squamous cell carcinoma (1), verrucous carcinoma (1).
RASSF1	125	1	9	11	Verrucous carcinoma (1).
MGMT	86	3	103	43	Squamous cell carcinoma (2); carcinoma (1).
CDH1	81	3	172	56	Tongue carcinoma (1); squamous cell carcinoma (1).
CDKN2B	42	1	96	9	Squamous cell carcinoma (1).

Table 6 shows that methylation frequency shown for APC, BRCA1, CDH1, DAPK1, and RARB with oral cancer. Highest methylation frequency was shown with CDH1.

Table 7. Diseases related to oral cancer.

S. N.	Related disease	Top affiliating genes (text searches by Pubmeth)
1	no subtype specified	CDKN2A.
2	squamous cell carcinoma	CDKN2A, MGMT, CDH1, CDKN2B.
3	verrucous carcinoma	CDKN2B, , RASSF1.
4	carcinoma	MGMT.
5	tongue carcinoma	CDH1.

Most of the genes for oral cancer are related to squamous cell carcinoma (Table 7).

Figure 3 shows the protein–protein interaction network.

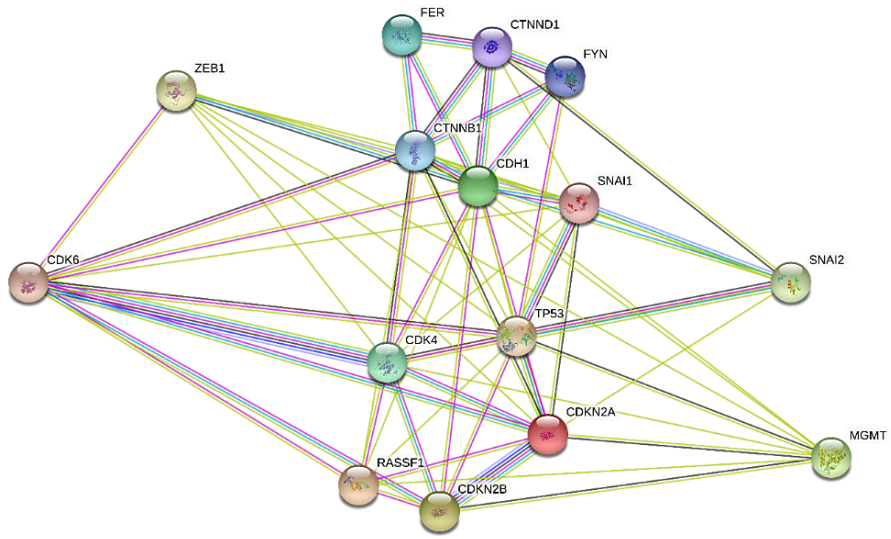

Figure 3. Protein–protein interaction for oral cancer causing genes.

Table 7 shows molecular diagnosis of CDH1 gene. The drugs Paclitaxel and Docetaxel show better activity against CDH1 gene.

Table 8. Activity of drugs against CDH1 gene as molecular therapy for oral cancer.

S. N.	Name of drug	Cdh1	
		Total energy in Kcal/mol	Active site
1	Cisplatin	–50.28	H-M-ILE-7 H-M-LEU-95 H-M-SER-156 H-S-ASN-157.
2	Carboplatin	–77.47	H-M-ALA-72 H-M-THR-73 H-S-THR-73 H-S-SER-156 V-S-GLN-41 V-M-THR-73 V-S-TRP-141 V-M-SER-155 V-M-SER-156.
3	5-fluorouracil	–71.39	H-M-ILE-7 H-M-LEU-95 H-M-THR-97 H-S-THR-97 H-S-SER-155 H-M-SER-156 H-S-ASN-157 V-M-THR-97 V-S-GLN-172.
4	*Paclitaxel*	*–118.76*	H-S-GLN-41 H-S-THR-45 V-M-GLY-40 V-M-GLN-41 V-S-GLN-41 V-S-THR-75 V-S-PHE-77 V-M-SER-158 V-M-PHE-159 V-S-PHE-159.
5	*Docetaxel*	*–126.47*	H-S-ASP-67 H-S-ASN-116 H-S-GLN-123 H-S-ARG-153 H-M-ASN-169 V-M-PRO-10 V-M-PRO-65 V-M-LEU-66 V-M-ASP-67 V-S-ASP-67 V-S-MET-119V-M-ARG-153 V-S-ARG-153.
6	Hydroxyurea	–55.85	H-M-ILE-7 H-M-LEU-95 H-M-THR-97 H-S-SER-155 H-M-SER-156 H-S-ASN-157 H-S-GLN-172.

Figure 4 shows the docking activities of drugs with CDH1.

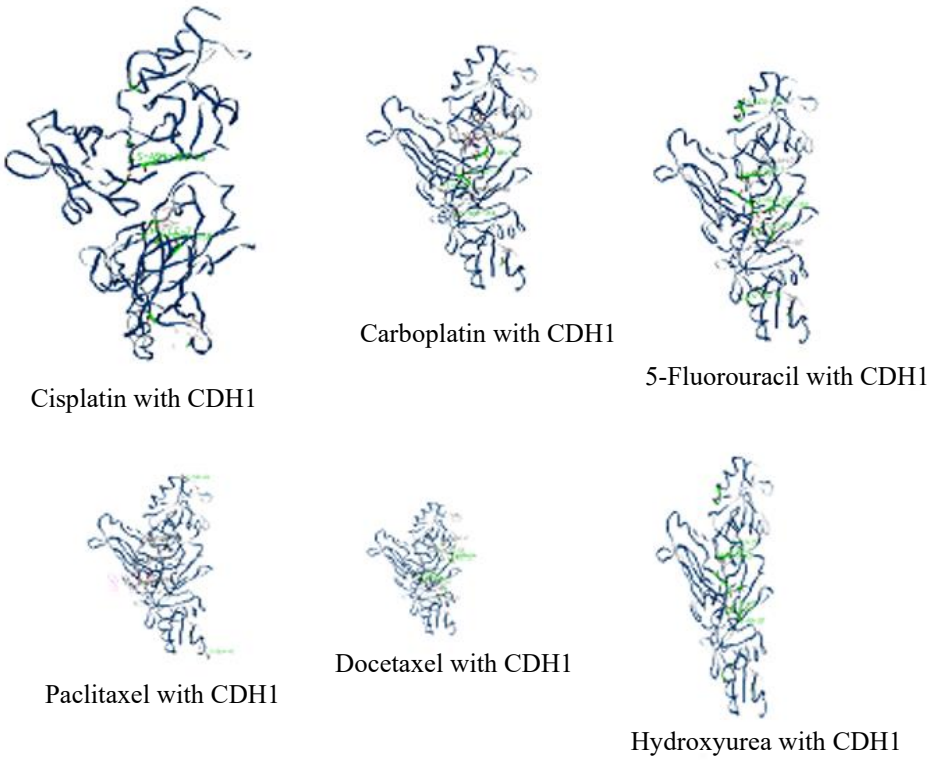

Figure 4. Docking activities of drugs with CDH1.

The work was confirmed that drugs Paclitaxel and Docetaxel show better activity against CDH1 gene and HPV16.

Oral cancer is an important health issue caused by sunlight exposure, smoking, and alcohol consumption [11]. Human papillomavirus (HPV) is the causal agent of cervical and oral cancer. Paclitaxel and docetaxel are researched as active compounds against a range of human cancers [6]. The present study shows that Paclitaxel and docetaxel are better molecular compounds for the control of HPV16 and oral cancers.

CONCLUSION

The gene prediction studies showed 7 genes from HPV16 strain. The drugs Paclitaxel and Docetaxel show better activity against HPV16. Methylation frequency is shown for APC, BRCA1, CDH1, DAPK1 and RARB with oral cancer. Highest methylation frequency was shown with CDH1. Protein alignment of CDH1, E6, and E7 by SPDBV have shown structural similarity. Methylation frequency has been analyzed for APC, BRCA1, CDH1, DAPK1 and RARB with oral cancer. Highest methylation frequency was shown with CDH1. The drugs Paclitaxel and Docetaxel have shown better activity against CDH1 gene. Further *in vivo* and *in vitro* analysis may provide better mechanism and treatment methods for oral cancer.

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Conflicts of Interest

There is no known conflict of interest associated with the publication.

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