International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

Investigation of Momordica charantia phytochemicals against PIM1 Kinase: A Computational Approach

Mudassir Alam[1*], Kashif Abbas[1], Mohd Mohsin[1], Imtiyaz Ali Zairy[2]
[1]Department of Zoology, Aligarh Muslim University, Aligarh, India. 202002
[2]Department of Biochemistry, Aligarh Muslim University, Aligarh, India. 202002

ABSTRACT: Prostate cancer is a significant contributor to male cancer-related mortality. PIM1 kinase has implications in the development and progression of various cancers, particularly prostate cancer. PIM1, a serine/threonine protein kinase plays a crucial role in cellular processes including survival, growth and differentiation. In prostate cancer increased PIM1 expression is associated with a more aggressive phenotype and poorer patient outcomes. It has emerged as a promising therapeutic target for prostate cancer treatment. The development of PIM1 kinase inhibitors has greatly enhanced and progressed significantly. Different stages of clinical trials demonstrating their potential as therapeutic agents. *Momordica charantia*, or bitter melon has a long history in traditional medicine for various health conditions, it is very rich in secondary metabolites like triterpenoids, glycosides, alkaloids, flavonoids and phenolic acids. Bitter melon is considered to have medicinal properties including potential anticancer phytochemicals. This study employs virtual screening, molecular dynamic simulation and ADME/T analysis to explore bitter melon's phytochemicals and their interaction with PIM1 kinase. The goal is to understand the molecular details and pharmacokinetics of bitter melon compounds evaluating their potential as therapeutic agents against prostate cancer. In our present study, it was found that out of all investigated phytochemicals catechin and gallic acid shows satisfactory result depending upon various parameters taken into consideration for conducting the study.

KEYWORDS: Prostate, cancer, PIM1 kinase, Momordica charantia, molecular docking, phytochemicals

1. INTRODUCTION

PIM1 kinase is a serine/threonine protein kinase belonging to the PIM kinase family, plays a pivotal role in regulating various cellular processes like cell survival, growth and differentiation through the phosphorylation of target proteins [1]. It is implicated in modulating signalling pathways activated by growth factors, cytokines and stress signals [2]. In the context of prostate cancer elevated PIM1 expression is linked to more aggressive tumor characteristics, potentially leading to poorer prognoses for prostate cancer patients [3]. Inhibitors of PIM1 kinase have been developed and are currently undergoing early-stage clinical trials for prostate cancer treatment. In preclinical studies, these inhibitors have demonstrated efficacy in impairing prostate cancer cell proliferation, survival, and angiogenesis leading to tumour regression [4]. Clinical trials play a fundamental role in advancing life-extending and curative interventions for cancer patients. They are essential for facilitating the transition of novel treatments, also generating essential data for regulatory approvals, enabling the integration of new drugs into widespread clinical practice. PIM1 kinase inhibitors exhibit anti-inflammatory and antioxidant properties further underscoring their potential as anticancer agents [5]. Several promising PIM1 kinase inhibitors, including small molecules like SGI-1776 and peptide inhibitors such as PIM447, are currently in various stages of clinical development [6][7]. However, it is crucial to emphasize that the use of PIM1 kinase inhibitors for prostate cancer therapy is still in the early stages necessitating further research to understand their mechanisms of action comprehensively and optimize their clinical applications. *Momordica charantia*, commonly known as bitter melon has long been employed in traditional medicine for centuries to address diverse health conditions including hepatitis [8] diabetes, obesity and various neurodegenerative disorders [9][10]. Bitter melon has recently garnered utmost attention for its potential source of natural compounds with anticancer properties. It contains several secondary metabolites such as triterpenoids, glycosides, alkaloids, flavonoids and phenolic acids which are essential in alleviating the broad range of diseases [11]. Studies have revealed that the phytochemicals present in bitter melon extract have great potency to inhibit cell proliferation, induce apoptosis and modulate the immune system, critical factors in managing prostate cancer [12][13]. Additionally, these phytochemicals also demonstrate anti-inflammatory, antioxidant and anti-angiogenic properties, further bolstering their potential as anticancer agent [14]. In this study,

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

various in-silico methods are employed to analyze the phytochemicals of bitter melon extract concerning their interaction with PIM1 kinase, specifically in the context of prostate cancer.

2. MATERIAL AND METHODS

2.1 Target protein preparation

The PDB website (https://www.rcsb.org/) was employed to obtain the pdb file of human Pim-1 kinase with the ID 6MT0. The Protein Data Bank (PDB) stores data on experimental protein and nucleic acid structures. To refine the protein for docking, water molecules were eliminated using PyMOL [15], an open-source molecular visualization software.

2.2 Ligand retrieval and preparation

Compound structures of bitter gourd were acquired in sdf file format from the PubChem database, which offers details on chemical compounds, including their structure, formula, and molecular weight (https://pubchem.ncbi.nlm.nih.gov/source/15751). Table 1 presents the compound information. Ligand preparation was conducted using the OpenBabel [16] tool from PyRx 0.8 [17], and the ligand energy was minimized using the mmff94 force field. The sdf file format of the ligands was converted to pdbqt format to render it executable.

Table1: Phytochemicals with their molecular weight and Pubchem ID.

Ligand	MW	PUBCHEM ID
Catechin	290.27	**9064**
Gallic acid	170.12	**370**
Karavilagenin A	486.80	**16079963**
Kuguacin J	454.70	**25243357**
Karaviloside II	648.90	**16093694**
Momordicoside K	648.90	**57330180**

2.3 Molecular Docking

A molecular docking research was performed utilising bitter gourd compounds as ligand groups and pim1 kinase as a macromolecule. The AutoDock Vina [18] tool from PyRx 0.8 was utilised to conduct the molecular docking investigation.

2.4 Visualization of Docking Results

After the docking simulation, the most favorable docked pose, determined by the best negative score (docking score), was selected as the optimal configuration for the respective chemical and protein. To analyze unbound interactions, Discovery Studio 4.5 [19] was employed for visualizing and presenting the top-docked position.

2.5 Analysis of protein-ligand interactions

The generation of Ligplot involved utilizing the Ligplot+ program to assess hydrophobic and hydrogen bond interactions between the ligand and the target protein [20]. Ligplot provides a two-dimensional representation of the interactions between the compound and the protein.

2.6 Prediction of Physiochemical Properties

The assessment of the drug-like characteristics of the compounds was conducted using the DruLito program. This investigation determined the count of rotatable bonds and compliance with Lipinski's Rule of 5 [21], which outlines the criteria for orally active drugs to maintain their pharmacological integrity.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

2.7 Prediction of absorption, metabolism and distribution

Absorption, distribution, and metabolism prediction of the chosen compound were done using admetSAR [22] (http://lmmd.ecust.edu.cn/admetsar2/).

2.8 Prediction of toxicity

ProTox-II (https://tox-new.charite.de/protox_II/index.php?site=compound_input) has been employed for toxicity prediction of chosen compound [23]. It is a web-based virtual toxicity laboratory for predicting several toxicological endpoints connected to a chemical structure. It is accessible to academic and non-commercial users. ProTox-II includes computer-based models trained on actual data (in vitro or in vivo) to forecast the hazardous potential of current and hypothetical substances.

2.9 Prediction of biological activity of the compound

To anticipate the biological activities of the selected molecules, the PASS web server (http://www.pharmaexpert.ru/passonline) was used [24]. Using multilayer atom neighbour descriptors, the PASS analysis assists in analysing the effects of a drug entirely based on its molecular formula, meaning that its biological behaviour is exclusively governed by its chemical structure.

3. RESULT AND DISCUSSION

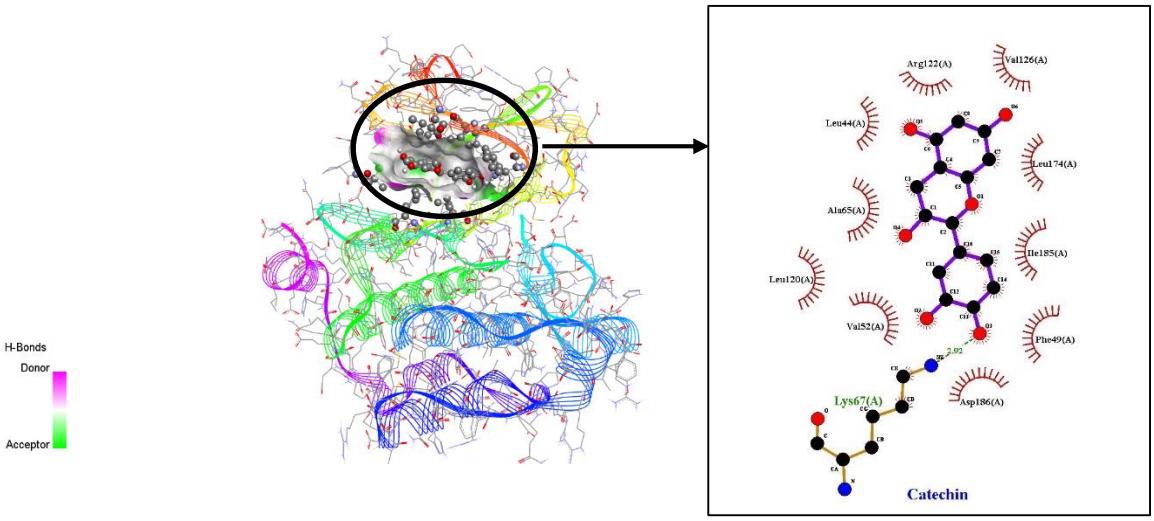
3.1 Docking score of the compounds

3D crystal structure of pim-1 kinase (PDB ID: 6MT0) was used for docking study. Autodock Vina from PyRx 0.8 was used for analysis. Protein was converted to macromolecule and all the selected compounds were first minimised with mmff94 forcefield and then finally converted to pdbqt format suing OpenBabel in PyRx. Blind docking was performed with grid box dimension (49.22 Å ×55.71 Å ×46.56 Å) and centre (-39.17, -13.23, -0.41). The exhaustiveness was set to 8 by default. Table 2 summarises the details of ligands or compounds with their docking score. Best docked poses and schematic 2D representation of their interaction with target protein is given in Figure 1 (a-f) . We observed that all of the chosen compound present in bitter gourd extract shown good docking score.

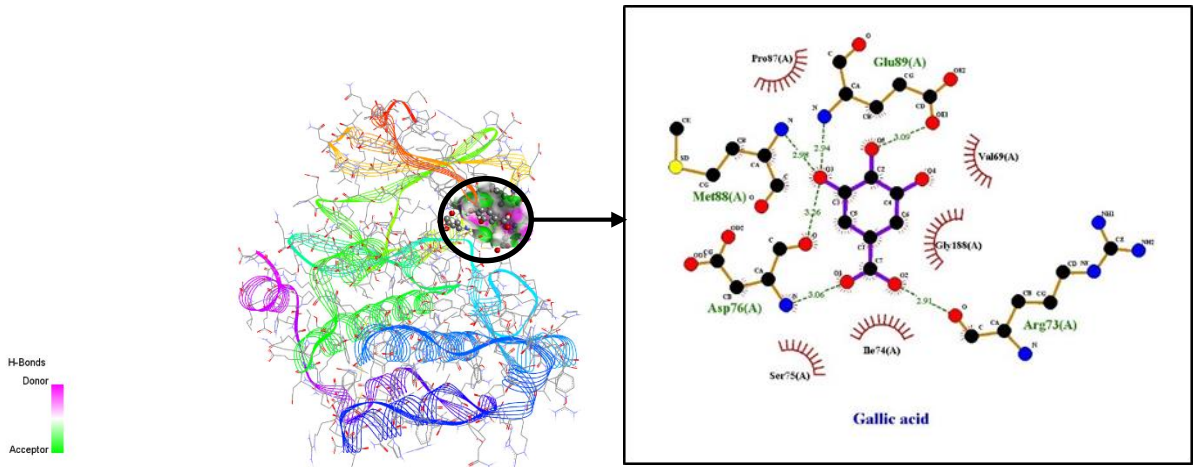
Table 2: Molecular details (MW and PUBCHEM ID) alongside docking scores (kcal/mol) for selected ligands. Lower scores, such as -8.7 for Momordicoside K, indicate strong binding affinity.

Ligand name	MW	PUBCHEM ID	Docking Score (kcal/mol)
Catechin	290.27	9064	**-8**
Gallic acid	170.12	370	**-6.2**
Karavilagenin A	486.80	16079963	**-8.2**
Kuguacin J	454.70	25243357	**-8.3**
Karaviloside II	648.90	16093694	**-7.4**
Momordicoside K	648.90	57330180	**-8.7**

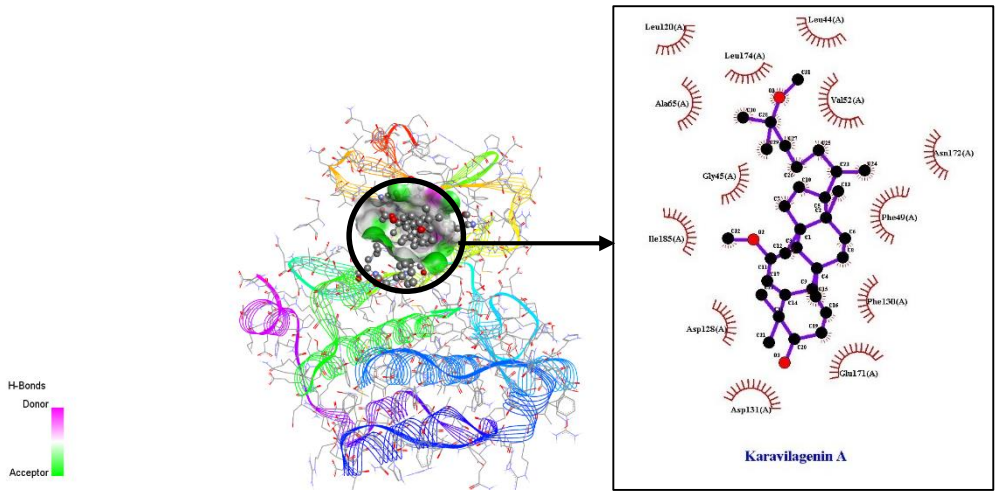
International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org



(a)

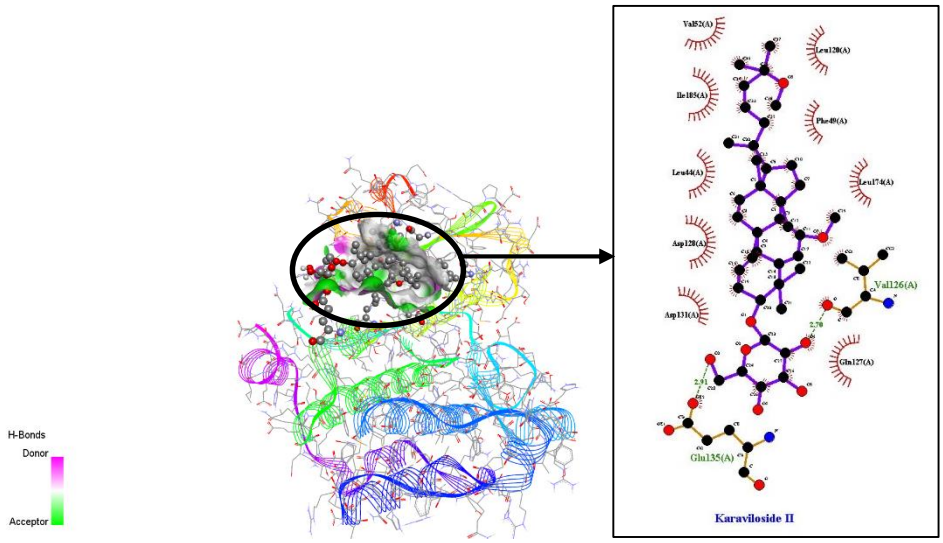


(b)

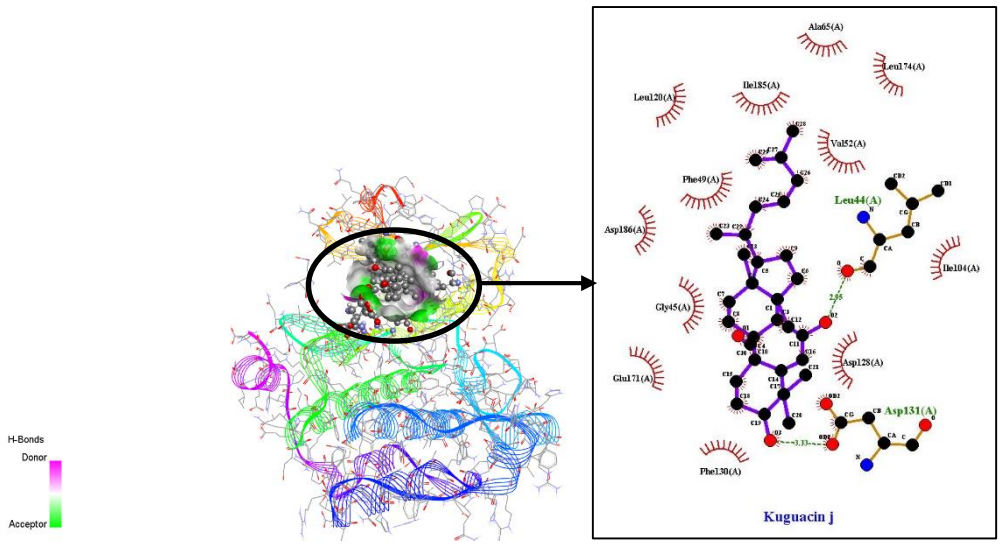


(c)

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

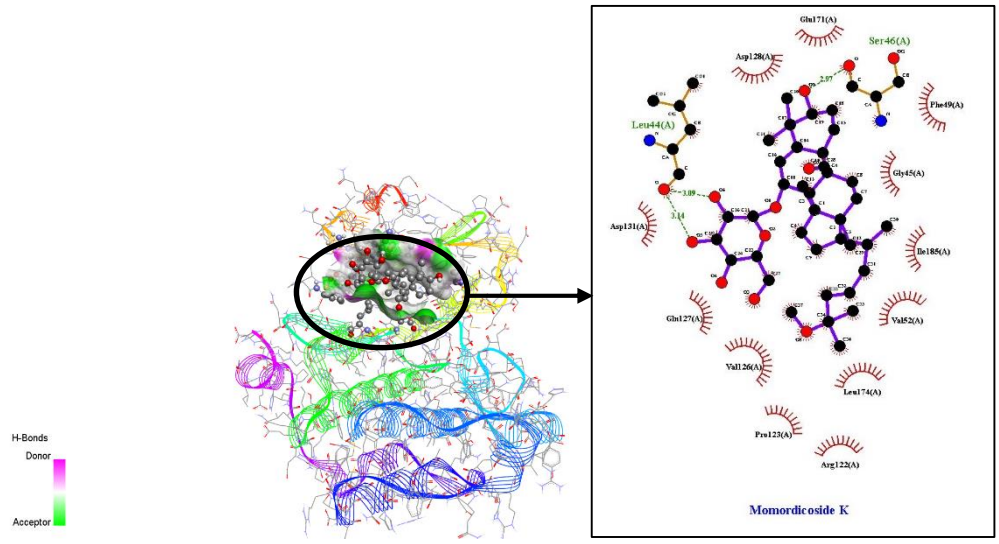


(d)



(e)

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org



(f)

Figure 1: 3D and 2D representation of protein ligand interaction: (a) Catechin (b) Gallic acid (c) Karavilagenin A (d) Karaviloside II (e) Kuguacin J (f) Momordicoside K

3.2 Protein-ligand interaction

Hydrophobic interactions and hydrogen bonding emphasise protein-ligand interactions. Summary of the interaction is given in Table 3 . The catechin molecule showed hydrophobic interaction with Leu44, Phe49, Val52, Ala65, Arg122, Val126, Leu174, Ile185, Asp186 while hydrogen bonding with Lys67. Gallic acid has shown hydrophobic interaction with amino acids such as Val69, Ile74, Ser75, Pro87, Gly188 while hydrogen bonding with Arg73, Asp76, Met88, Gly89. The molecule Karavilagenin A exhibit hydrophobic interaction with Leu44, Gly45,Phe49, Val52, Ala65, Leu120, Asp128, Phe130, Asp131, Glu171, Asn172, Leu174, Ile185, and no hydrogen bonding with any amino acids. Furthermore, karaviloside II has shown hydrophobic interaction with Leu44, Phe49, Val52, Leu120, Gln127, Asp128, Asp131, Leu174, Ile185 and hydrogen bonding with Val126, Glu135. Next molecule that is kuguacin J exhibit hydrophobic interaction with amino acids namely Gly45, Phe49, Val52, Ala65, Ile104, Leu120, Asp128, Phe130, Glu171, Leu174, Ile185, Asp186 and hydrogen bond interaction with Leu44, Asp131. Last, momordicoside K has shown hydrophobic interaction with Gly45, Phe49, Val52, Arg122, Pro123, Val126, Gln127, Asp128, Asp131, Glu171, Leu174, Ile185 and hydrogen bonding with amino acid Leu44 and Ser46.

Table 3: Summary of hydrophobic and hydrogen bond amino acids interaction between ligand and protein

Ligand name	Hydrophobic interaction	Hydrogen bonding
Catechin	Leu44, Phe49, Val52, Ala65, Arg122, Val126, Leu174, Ile185, Asp186	Lys67
Gallic acid	Val69, Ile74, Ser75, Pro87, Gly188	Arg73, Asp76, Met88, Gly89
Karavilagenin A	Leu44, Gly45,Phe49, Val52, Ala65, Leu120, Asp128, Phe130, Asp131, Glu171, Asn172, Leu174, Ile185,	----------

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

Kuguacin J	Gly45, Phe49, Val52, Ala65, Ile104, Leu120, Asp128, Phe130, Glu171, Leu174, Ile185, Asp186	Leu44, Asp131
Karaviloside II	Leu44, Phe49, Val52, Leu120, Gln127, Asp128, Asp131, Leu174, Ile185	Val126, Glu135
Momordicoside K	Gly45, Phe49, Val52, Arg122, Pro123, Val126, Gln127, Asp128, Asp131, Glu171, Leu174, Ile185	Leu44, Ser46

3.3 Pharmacokinetics and Toxicological Properties Analysis

To assess drug similarity, it is essential to determine the ADME/T characteristics of ligands. The DruLito program was employed to evaluate the pharmacological properties of compounds present in bitter gourd extract. Various drug similarity rules, including Lipinski's rule, MDDR-like rule, Ghose filter, BBB similarity, CMC-50-like rule, unweighted QED, Veber filter, and weighted QED, were applied for compound identification. The findings are consolidated in Table 4. Additionally, Table 5 presents the predicted absorption, distribution, and metabolism of selected compounds using the admetSAR server. All molecules exhibited positive human intestinal absorption, with Molecules 1, 3, and 5 demonstrating blood-brain permeability. Tables 4 and 5 provide a summary of the results.

Table 4: Pharmacological properties of the selected compounds evaluated using DruLito server.

Name	P. ID	MW	logp	Alogp	HBA	HBD	TPSA	nRB
Catechin	9064	290.08	0.852	0.936	6	5	110.38	1
Gallic acid	370	170.02	0.964	0.721	5	4	97.99	1
Karavilagenin A	16079963	486.41	8.509	2.71	3	1	38.69	6
Karaviloside II	16093694	648.46	7.401	0.963	8	4	117.84	9
Kuguacin J	25243357	454.34	7.409	2.079	3	2	57.53	5
Momordicoside K	57330180	648.42	5.224	0.279	9	5	145.91	9

Table 5: Absorption, distribution, and metabolism of the chosen compound as per admetSAR online toolkit

Parameters	Catechin (1)	Gallic acid (2)	Karavilagenin A (3)	Karaviloside II (4)	Kuguacin J (5)	Momordicoside K (6)
ABSORPTION						
BB-barrier	+	-	+	-	+	-
Human intestinal absorption	+	+	+	+	+	+
P-glycoprotein substrate	-	-	+	-	+	+
P-glycoprotein inhibitior	-	-	+	+	-	+

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

DISTRIBUTION						
Subcellular localization	Mitochondria	Mitochondria	Mitochondria	Mitochondria	Mitochondria	Mitochondria
METABOLISM						
CYP2C9 substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate
CYP2D6 substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate	Non-substrate
CYP3A4 substrate	Non-substrate	Non-substrate	Substrate	Substrate	Substrate	Non-substrate
CYP1A2 inhibition	Inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor
CYP2C9 inhibition	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor
CYP2D6 inhibition	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor
CYP2C19 inhibition	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor	Non-inhibitor

Protox II server was used for toxicity prediction of the compounds. Table 6 summarises the result. All of the chosen molecule were safe and shown negligible or no sign of any toxicity as per Protox II server.

Table 6: Toxicological prediction of the compounds using PROTOX- II server

Parameters	**Catechin**	**Gallic acid**	**Karavilagenin A**	**Karaviloside II**	**Kuguacin J**	**Momordicoside K**
Carcinogenicity	NO	NO	NO	NO	NO	NO
Mutagenicity	NO	NO	NO	NO	NO	NO
Cytotoxicity	NO	NO	NO	NO	NO	NO
Immunotoxicity	Weak/low	NO	Weak/low	Weak/low	Weak/low	Weak/low
Hepatotoxicity	Weak/low	NO	Weak/low	NO	NO	Weak/low

3.4 Predictions of Biological Activity of Compounds

The verification of the anticipated biological activity was carried out using the PASS webserver, and the chosen compounds were found to exhibit identical biological activities as predicted. This study showed that the molecules in series 1–6 are anticarcinogenic, antineoplastic, treat prostate cancer, and stop pim1 kinase from working (molecules 1 and 2). Pa ranges from 0.24 to 0.79 for anticarcinogenic, and it goes from 0.31 to 0.88 for antineoplastic. The Pa value for treating prostate cancer was between 0.18 and 0.42. When Pa > Pi, the above Pa values show that the molecules are likely to have strong anticancer properties. The prediction is summed up in the table 7.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

Table 7: Biological activity prediction of compounds (Pa = probability to be active; Pi = probability to be inactive

Compound name	Biological activity	Pa	Pi
CATECHIN	Anticarcinogenic	0.795	0.005
	Antineoplastic	0.675	0.030
	Pim1 kinase inhibitor	0.154	0.075
	Prostate cancer treatment	0.426	0.018
GALLIC ACID	Anticarcinogenic	0.395	0.031
	Antineoplastic	0.313	0.145
	Pim1 kinase inhibitor	0.507	0.003
	Prostate cancer treatment	0.183	0.085
KARAVILAGENIN A	Anticarcinogenic	0.240	0.088
	Antineoplastic	0.841	0.008
	Pim1 kinase inhibitor	--	--
	Prostate cancer treatment	0.270	0.049
KUGUACIN J	Anticarcinogenic	0.331	0.048
	Antineoplastic	0.874	0.005
	Pim1 kinase inhibitor	--	--
	Prostate cancer treatment	0.381	0.025
KARAVILOSIDE II	Anticarcinogenic	0.605	0.012
	Antineoplastic	0.862	0.006
	Pim1 kinase inhibitor	--	--
	Prostate cancer treatment	0.228	0.063
MOMORDICOSIDE K	Anticarcinogenic	0.498	0.019
	Antineoplastic	0.884	0.005
	Pim1 kinase inhibitor	--	--
	Prostate cancer treatment	0.240	0.059

4. CONCLUSION

M. charantia has long been utilised as an herbal treatment with strong pharmacological benefits. Research has explored its medicinal attributes like antibacterial, antiviral, antitumor, immunomodulatory, antioxidant, anthelmintic, antimutagenic, antilipolytic, antifertility, hepatoprotective, anti-inflammatory, anti-ulcerogenic, antioxidative and immune-modulatory properties. PIM1 has emerged as a key player in prostate cancer (PCa) carcinogenesis, where its overexpression enhances the tumorigenicity of prostate

cancer. Current approaches to inhibit PIM in cancer treatment have predominantly centered on a monotherapeutic strategy. This typically involves the use of ATP (adenosine triphosphate)-competitive drugs targeting the kinase activity of the protein, preventing its phosphorylation of downstream effectors. These efforts utilize compounds like quinones or other classes of small molecule inhibitors. A paradigm change including computer-based simulations and data analysis provides vital insight into the complexities of proteins and ligands. The research concentrated on computational approaches for determining the therapeutic capabilities of phytochemicals found in *M. charantia* against PIM1 kinase. In this research, several in silico methodologies were used, and the results were reviewed. Based on the numerous parameters utilised in our research, we conclude that catechin and gallic acid has shown good biological activity of being PIM1 kinase inhibitor and also exhibited satisfactory docking scores, ADME, and toxicological measures. Further efficacy of these phytochemicals might be confirmed by using in vitro and in vivo methods.

CONFLICT OF INTEREST: Authors declare no conflict of interest.

REFERENCES

1. Tursynbay, Y., Zhang, J., Li, Z., Tokay, T., Zhumadilov, Z., Wu, D., et al. (2016). Pim-1 kinase as cancer drug target: An update. Biomedical reports, 4(2), 140–146. https://doi.org/10.3892/br.2015.561
2. Warfel, N. A., & Kraft, A. S. (2015). PIM kinase (and Akt) biology and signaling in tumors. Pharmacology & therapeutics, 151, 41–49. https://doi.org/10.1016/j.pharmthera.2015.03.001
3. Narlik-Grassow, M., Blanco-Aparicio, C. and Carnero, A. (2014), The PIM Family of Serine/Threonine Kinases in Cancer. Med. Res. Rev., 34: 136-159. https://doi.org/10.1002/med.21284
4. Holder, S. L., & Abdulkadir, S. A. (2014). PIM1 kinase as a target in prostate cancer: roles in tumorigenesis, castration resistance, and docetaxel resistance. Current cancer drug targets, 14(2), 105–114. https://doi.org/10.2174/1568009613666131126113854
5. Luszczak, S., Kumar, C., Sathyadevan, V. K., Simpson, B. S., Gately, K. A., et al. (2020). PIM kinase inhibition: co-targeted therapeutic approaches in prostate cancer. Signal transduction and targeted therapy, 5(1), 7. https://doi.org/10.1038/s41392-020-0109-y
6. Cervantes-Gomez, F., Chen, L. S., Orlowski, R. Z., & Gandhi, V. (2013). Biological effects of the Pim kinase inhibitor, SGI-1776, in multiple myeloma. Clinical lymphoma, myeloma & leukemia, 13 Suppl 2(0 2), S317–S329. https://doi.org/10.1016/j.clml.2013.05.019
7. Peters, T. L., Li, L., Tula-Sanchez, A. A., Pongtornpipat, P., et al. (2016). Control of translational activation by PIM kinase in activated B-cell diffuse large B-cell lymphoma confers sensitivity to inhibition by PIM447. Oncotarget, 7(39), 63362–63373. https://doi.org/10.18632/oncotarget.11457
8. Alam, M., Abbas, K., Ali, M., Kamal, S., & Bhagia, S. (2023). An in-silico study encompassing virtual screening and ADME/T properties of phytochemicals present in leaf extract of Trapa natans in relevance to sodium taurocholate co-transporting polypeptide involved in hepatitis B. Journal of Pharmacognosy and Phytochemistry, 12(1), 641-652.
9. Alam, M., & Abbas, K. (2021). An insight into neurodegenerative disorders, their therapeutic approaches and drugs available for tackling with neurodegeneration: a review. IAR Journal of Medical Case Reports, 2(3).
10. Abbas, K., Alam, M., Ali, V., & Ajaz, M. (2023). Virtual screening, molecular docking and ADME/T properties analysis of neuroprotective property present in root extract of chinese skullcap against β-site amyloid precursor protein cleaving enzyme 1 (BACE1) in case of alzheimer's disease. Journal of Pharmacognosy and Phytochemistry, 12(1), 146-153.
11. Joseph, B., & Jini, D. (2013). Antidiabetic effects of Momordica charantia (bitter melon) and its medicinal potency. Asian Pacific Journal of Tropical Disease, 3(2), 93–102. https://doi.org/10.1016/S2222-1808(13)60052-3
12. Li, C. J., Tsang, S. F., Tsai, C. H., Tsai, H. Y., Chyuan, J. H., & Hsu, H. Y. (2012). Momordica charantia Extract Induces Apoptosis in Human Cancer Cells through Caspase- and Mitochondria-Dependent Pathways. Evidence-based complementary and alternative medicine : eCAM, 2012, 261971. https://doi.org/10.1155/2012/261971
13. Jia, S., Shen, M., Zhang, F., & Xie, J. (2017). Recent Advances in Momordica charantia: Functional Components and Biological Activities. International journal of molecular sciences, 18(12), 2555. https://doi.org/10.3390/ijms18122555

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 07 Issue 01 January 2024
DOI: 10.47191/ijcsrr/V7-i1-01, Impact Factor: 6.789
IJCSRR @ 2024
www.ijcsrr.org

14. Raina, K., Kumar, D., & Agarwal, R. (2016). Promise of bitter melon (Momordica charantia) bioactives in cancer prevention and therapy. Seminars in cancer biology, 40-41, 116–129. https://doi.org/10.1016/j.semcancer.2016.07.002
15. Schrödinger, L., & DeLano, W. (2020). PyMOL. Retrieved from http://www.pymol.org/pymol
16. O'Boyle, N. M. (2011). Banck M. James CA Morley C. Vandermeersch T. Hutchison GR Open Babel: An open chemical toolbox. J. Cheminf, 3(1), 33.
17. Dallakyan, Sargis & Olson, Arthur. (2015). Small-Molecule Library Screening by Docking with PyRx. Methods in molecular biology (Clifton, N.J.). 1263. 243-250. 10.1007/978-1-4939-2269-7_19.
18. Trott, O., & Olson, A. J. (2010). AutoDock Vina: improving the speed and accuracy of docking with a new scoring function, efficient optimization, and multithreading. Journal of computational chemistry, 31(2), 455-461.
19. Biovia, D.S. (2021) Discovery Studio Modeling Environment. Dassault Syst. Release, San Diego, 4.
20. Laskowski R A, Swindells M B (2011). LigPlot+: multiple ligand-protein interaction diagrams for drug discovery. J. Chem. Inf. Model., 51, 2778-2786. [PubMed id: 21919503]
21. Lipinski, C. A., Lombardo, F., Dominy, B. W., & Feeney, P. J. (1997). In vitro models for selection of development candidatesexperimental and computational approaches to estimate solubility and permeability in drug discovery and development settings. Adv Drug Deliv Rev, 23(1), 3-25.
22. Cheng, F., Li, W., Zhou, Y., Shen, J., Wu, Z., Liu, G., ... & Tang, Y. (2012). admetSAR: a comprehensive source and free tool for assessment of chemical ADMET properties.
23. Banerjee, P., & Eckert, O. (2018). A.; Schrey, AK; Preissner, R. ProTox-II: A webserver for the prediction of toxicity of chemicals. Nucleic Acids Res, 46, W257-W263.
24. Filimonov D.A., Lagunin A.A., Gloriozova T.A., Rudik A.V., Druzhilovskii D.S., Pogodin P.V., Poroikov V.V. (2014). Prediction of the biological activity spectra of organic compounds using the PASS online web resource. Chemistry of Heterocyclic Compounds, 50 (3), 444-457.

Cite this Article: Mudassir Alam, Kashif Abbas, Mohd Mohsin, Imtiyaz Ali Zairy (2024). Investigation of Momordica charantia phytochemicals against PIM1 Kinase: A Computational Approach. International Journal of Current Science Research and Review, 7(1), 01-11