INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

Research Article

An Approach Of Computer Aided Drug Design (CADD) Tools For In-silico Evaluation Of Various Derivatives Of Antidiabetic Standard Drugs

Anushree Suga[1], Ashwini Paschapur[1], Somashekhar M Metri*, Trupti A Hunnura[2], Hanamant B Sannakki[2]

Department of Pharmaceutical Chemistry, BLDEA's SSM College of Pharmacy and Research Centre Vijayapura-586103, Karnataka, India.

ARTICLE INFO

Published: 19 Oct 2024
Keywords:
Insilco study, ADME/T study, Pass Prediction, Antidiabetic Activity, Antitubercular Activity
DOI:
10.5281/zenodo.13955358

ABSTRACT

An in-silico ADME/T study utilizing Swiss ADME software was conducted on novel derivatives of standard antidiabetic drugs, revealing reduced toxicity levels compared to existing medications. Docking scores showed significant interactions with antidiabetic and antitubercular properties, successfully binding to protein PDB IDs 8Q0T and 1H5U. Predictive analysis yielded Pa and Pi values (0-1), indicating promising antidiabetic activity. These findings suggest the derivatives' potential for development into enhanced efficacy antidiabetic compounds, opening new avenues for antidiabetic drug discovery and providing valuable leads for future research in combating diabetes.

INTRODUCTION

In chemistry, "docking" often refers to a computational method used in molecular modeling to predict how small molecules, such as drugs, interact with larger molecules, such as proteins or enzymes. This technique is instrumental in drug discovery and design.1 Here's a breakdown of how docking works: The process involves placing a small molecule (the ligand) into the requisite site of a larger fragment (the target, usually a protein) to predict how well they fit together. The goal is to find the optimal orientation and conformation of the ligand that maximizes its interaction with the target.2 In the context of chemistry, particularly in drug development and pharmaceuticals, ADME, and toxicity are crucial for understanding how compounds behave in biological systems.3 Refers to the adverse effects a compound can have on an organism. It involves studying the harmful effects that can occur at various concentrations and understanding the safety margin between

***Corresponding Author:** Somashekhar M Metri
Address: *Department of Pharmaceutical Chemistry, BLDEA's SSM College of Pharmacy and Research Centre Vijayapura-586103, Karnataka, India.*
Email ✉: csm.som@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

therapeutic and toxic doses.4 Together, ADME and toxicity studies help predict a compound's behavior in the body, its effectiveness, and its safety profile, guiding the development of new drugs and chemicals.5 Numerous physiological functions were predicted using the online communication PASS (prediction of activity profiles for compounds; http://www.pharmaexpert.ru/ PASS online/ index.php). According to Kumaresan et al. (2015), this instrument was developed to predict a variety of biological processes with 95% accuracy.6 The structures were created in ChemDraw 16.0, translated to Smiles format, and used with the PASS online version to predict the biological spectrum (Kawsar et al. 2022a). The outcome was displayed as the likelihood of the active substance, Pa, and the inactive substance, Pi. In the range of 0.000–1.000, Pa > Pi is taken into consideration here, and Pa + Pi ≠ 1 generally.7 The PASS prediction results were interpreted and applied with flexibility: [i] the prospect of discovery of the movement empirically is in elevation when Pa > 0.5; [ii] the possibility of conclusion the action empirically is lesser if 0.5 < Pa < 0.7, but the composite is probable not as comparable to recognized remedial products agents, and [iii] the prospect of conclusion the activity through researches is lesser but not the structure-based prospect (Filimonov et al. 2014). Therefore, the chemical's intrinsic feature is the spectrum's expected activity.8

MATERIALS AND METHODS:

The docking investigations are performed to evaluate the different forms of bimolecular connections and ligand-receptor attachment intensities. PyRX, PyMOL, Biovia Discovery Studios 2020, Auto dock Vina, and other tools were used for these docking studies. Proteins were used in the docking investigation, specifically Polyketide synthase, an antitubercular amino acid, and its structure in crystals. PKS13 (PDB ID: 8Q0U) The enzyme glycogen phosphorylase is an anti-diabetic protein (PDB ID: 1H5U). An AMD Ryzen 7 3700U processor powered an HP 15s-eq0132au computer, which was utilized for the statistical task.

Proteins assembly

The RCSB Proteins The information a bank provided the crystalline structure of the antitubercular amino acids Polyketide synthesis the PKS13 (PDB ID: 8Q0U) as well as the anti-diabetic amino acids Glycogen phosphorylase (PDB ID: 1H5U). The amino acids were generated using the Swiss PDB viewers to remove the remaining agonists, and the resulting amino acids have been retained in the form of a PDB.

The Ligand setup

The three-dimensional models for the molecules were generated utilizing a chem sketch and submitted in BIOVIA Discovery Studio Visualizer-2020. A clustered sdf file had been generated after ligands reduction was completed utilizing the "A SMALL MOLECULES" procedure in BIOVIA Discovery Studios the Visualizer feature-2020.

Docking studies

Docking investigations become more important to minimize mistakes and determine the proper position of compounds inside the protein's active region. PyRx-Virtual Evaluation Tools was utilized to do the docking process. In the PyRx-Virtual Screens Instrument change ligands to pdbqt, then choose those as ligands in the Vina tutorial. ready-to-use proteins have been imported into the PyRx-Virtual Screening Tool and marked for macro molecule selection. The calculation of the binding-related amino acids and connection energy—the relationship between the substance that binds and the receptor was done.

Research on Drugs Likelihood

DruLiTO was used to import the chosen phytonutrients in sdf file and perform the therapeutic likelihood testing ADME/T Studies.

The Swiss ADME/T was used to download the SMILES of the chosen compounds while saving their ADME/T attributes. Scores have been summarized as shown in the table.

Predictions for PASS

The shortlisted compounds' SMILES were entered into the validation phase of the digital way2drug program.

RESULTS AND DISCUSSION

The following are the smiles of some standard antidiabetic drugs with their derivatives:

Standard Drugs name with smiles	Derivative smiles
O=S(=O)(NC(=O)NCCC)c1ccc(Cl)cc1 **Chlorpropamide**	CCCN(CCC)C(=O)NS(=O)(=O)c1ccc(Cl)cc1 Derivative I
	CCCN(C(=O)NCCC)S(=O)(=O)c1ccc(Cl)cc1 Derivative II
O=S(=O)(NC(=O)NC1CCCCC1)c1ccc(cc1)CCNC(=O)c1ncc(C)nc1 **Glipizide**	O=S(=O)(NC(=O)N(C1CCCCC1)C1CCCCC1)c1ccc(CCNC(=O)c2ncc(C)nc2)cc1 Derivative I
	O=S(=O)(NC(=O)NC1CCCCC1)c1ccc(cc1)CCNC(=O)c1ncc(nc1)CC Derivative II
O=C(O)c1ccc(CC(=O)NC(CC(C)C)c2ccccc2N2CCCCC2)cc1OCC **Repaglinide**	COC(=O)c1ccc(CC(=O)NC(CC(C)C)c2ccccc2N2CCCCC2)cc1OCC Derivative I
	O=C(O)c1ccc(CC(=O)NC(CC(C)C)c2ccccc2N2CCCCC2)cc1OCOC Derivative II
N\C(=N/C(=N)N)N(C)C **Metformin**	N=C(\N=C(/N)N(C)C)N(C)C Derivative I
	N\C(=N/C(=N)N)N(C)OC Derivative II
O=C1SC(Cc2ccc(cc2)OCCc2ccc(CC)cn2)C(=O)N1 **Pioglitazone**	O=C1SC(Cc2ccc(cc2)OCCc2ccc(COC)cn2)C(=O)N1 Derivative I
	O=C1SC(Cc2ccc(cc2)OCCc2ccc(C)cn2)C(=O)N1 Derivative II
OC1OC(CO)C(OC2OC(CO)C(OC3OC(C)C(NC4C=C(CO)C(O)C(O)C4O)C(O)C3O)C(O)C2O)C(O)C1O **Acarbose**	OC1OC(CO)C(OC2OC(CO)C(OC3OC(OC)C(NC4C=C(CO)C(O)C(O)C4O)C(O)C3O)C(O)C2O)C(O)C1O Derivative I
	OC1OC(CO)C(OC2OC(CO)C(OC3OC(CC)C(NC4C=C(CO)C(O)C(O)C4O)C(O)C3O)C(O)C2O)C(O)C1O Derivative II

ADMET PREDICTION RESULTS:

Physicochemical Properties Chlorpropamide		Chlorpropamide d-I	Chlorpropamide d-II	Glipizide	Glipizide d-I	Glipizide d-II
Formula	C10H13ClN2O3S	C13H19ClN2O3S	C13H19ClN2O3S	C21H27N5O4S	C27H37N5O4S	C22H29N5O4S
Molecular weight	276.74 g/mol	318.82 g/mol	318.82 g/mol	445.54 g/mol	527.68 g/mol	459.56 g/mol
Molar Refractivity	65.16	79.67	79.67	115.30	142.12	120.11
TPSA	83.65 Å²	74.86 Å²	74.86 Å²	138.53 Å²	129.74 Å²	138.53 Å²
Lipophilicity						
Log $P_{o/w}$ (iLOGP)	1.28	2.68	3.24	2.51	3.35	2.28
Consensus Log $P_{o/w}$	1.77	2.66	2.77	1.97	3.43	2.19
Water Solubility						
Solubility	3.90e-01 mg/ml; 1.41e-03 mol/l	2.05e-01 mg/ml; 6.42e-04 mol/l	1.10e-01 mg/ml; 3.44e-04 mol/l	1.65e-01 mg/ml; 3.70e-04 mol/l	9.69e-03 mg/ml; 1.84e-05 mol/l	8.86e-02 mg/ml; 1.93e-04 mol/
Pharmacokinetics						
GI absorption	High	High	High	Low	Low	Low
Log K_p (skin permeation)	-6.38 cm/s	-6.35 cm/s	-6.04 cm/s	-7.66 cm/s	-7.14 cm/s	-7.44 cm/s
Druglikeness						
Lipinski	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	Yes; 1 violation: MW>500	Yes; 0 violation
Bioavailability Score	0.55	0.55	0.55	0.55	0.55	0.55
Medicinal Chemistry						
Synthetic accessibility	2.37	2.65	2.82	3.33	3.95	3.52

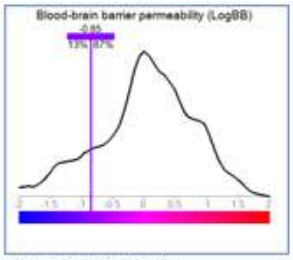
Physicochemical Properties Repaglinide		Repaglinide d-I	Repaglinide d-II	Metformin	Metformin d-I	Metformin d-II
Formula	C27H36N2O4	C28H38N2O4	C27H36N2O5	C4H11N5	C6H15N5	C4H11N5O
Molecular weight	452.59 g/mol	466.61 g/mol	468.59 g/mol	129.16 g/mol	157.22 g/mol	145.16 g/mol
Molar Refractivity	135.45	139.77	136.53	36.93	46.73	38.01
TPSA	78.87 Å²	67.87 Å²	88.10 Å²	91.49 Å²	68.71 Å²	100.72 Å²
Lipophilicity						
Log $P_{o/w}$ (iLOGP)	3.74	4.75	3.88	0.64	1.28	0.50
Consensus Log $P_{o/w}$	4.40	4.84	4.09	-0.92	-0.34	-1.01
Water Solubility						
Solubility	3.60e-05 mg/ml; 7.96e-08 mol/l	7.62e-06 mg/ml; 1.63e-08 mol/l	2.83e-03 mg/ml; 6.03e-06 mol/l	9.05e+01 mg/ml; 7.00e-01 mol/l	8.29e+01 mg/ml; 5.27e-01 mol/l	2.48e+02 mg/ml; 1.71e+00 mol/l
Pharmacokinetics						
GI absorption	High	High	High	High	High	High
Log K_p (skin permeation)	-5.38 cm/s	-5.23 cm/s	-5.77 cm/s	-7.99cm/s	-7.75cm/s	-8.06 cm/s
Druglikeness						
Lipinski	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation
Bioavailability Score	0.56	0.55	0.56	0.55	0.55	0.55
Medicinal Chemistry						
Synthetic accessibility	3.89	4.05	4.02	3.02	3.20	3.27

Physicochemical Properties Pioglitazone	Pioglitazone d-I	Pioglitazone d-II	Acarbose	Acarbose d-I	Acarbose d-II
Formula	C19H20N2O3S	C19H20N2O4S	C18H18N2O3S	C25H43NO18	C25H43NO19
Molecular weight	356.44 g/mol	372.44 g/mol	342.41 g/mol	645.60 g/mol	661.60 g/mol
Molar Refractivity	102.17	103.25	97.36	136.69	137.77
TPSA	93.59 Å²	102.82 Å²	93.59 Å²	321.17 Å²	330.40 Å²
Lipophilicity					
Log $P_{o/w}$ (iLOGP)	2.61	2.45	2.47	0.63	0.50
Consensus Log $P_{o/w}$	3.09	2.46	2.08	-6.22	-6.56
Water Solubility					

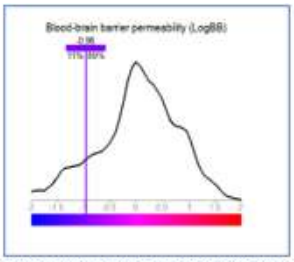
Solubility	1.76e-02 mg/ml ; 4.95e-05 mol/l	1.14e-04 mg/ml ; 3.07e-07 mol/l	3.21e-02 mg/ml ; 9.36e-05 mol/l	8.61e+04 mg/ml ; 1.33e+02 mol/l	4.15e+05 mg/ml ; 6.28e+02 mol/l
Pharmacokinetics					
GI absorption	High	High	High	Low	Low
Log K_p (skin permeation)	-5.81 cm/s	-6.72 cm/s	- 6.03 cm/s	-16.29 cm/s	-16.69cm/s
Drug likeness					
Lipinski	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation	No; 3 violations: MW>500, N or O>10, NH or OH>5	No; 3 violations: MW>500, N or O>10, NH or OH>5
Bioavailability Score	0.56	0.55	0.56	0.17	0.17
Medicinal Chemistry					
Synthetic accessibility	3.46	3.49	3.31	7.34	7.3

Physicochemical Properties Dapagliflozin		**Dapagliflozin d-II**	**Physicochemical Properties Dapagliflozin**
Formula	C21H25ClO6	C21H25ClO5	C20H23ClO6
Molecular weight	408.87 g/mol	392.87 g/mol	394.85 g/mol
Molar Refractivity	104.82	103.30	100.02
Lipophilicity			
Log $P_{o/w}$ (iLOGP)	3.17	2.76	2.47
Consensus Log $P_{o/w}$	2.18	2.42	1.76
Water Solubility			
Solubility	6.84e-02 mg/ml ; 1.67e-04 mol/l	3.54e-02 mg/ml ; 9.02e-05 mol/l	1.15e-01 mg/ml ; 2.92e-04 mol/l
Pharmacokinetics			
GI absorption	High	High	High
Log K_p (skin permeation)	-7.13 cm/s	-6.70 cm/s	- 7.30 cm/s
Drug likeness			
Lipinski	Yes; 0 violation	Yes; 0 violation	Yes; 0 violation
Bioavailability Score	0.55	0.55	0.55
Medicinal Chemistry			
Synthetic accessibility	4.52	4.09	4.41

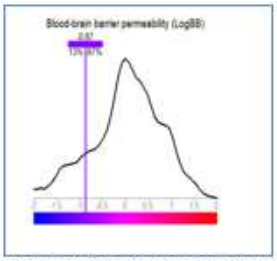
PREDICTION RESULTS:



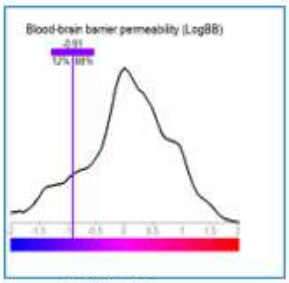
CHLORPROPAMIDE



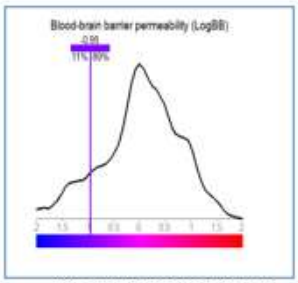
CHLORPROPAMIDE DERIVATIVE I



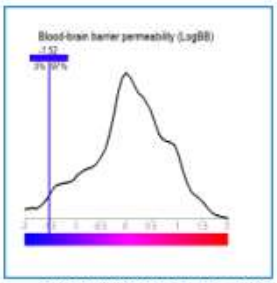
CHLORPROPAMIDE DERIVATIVE II



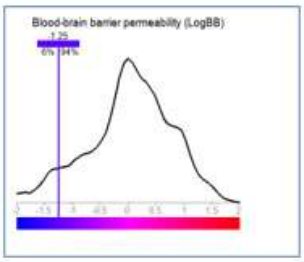
GLIPIZIDE



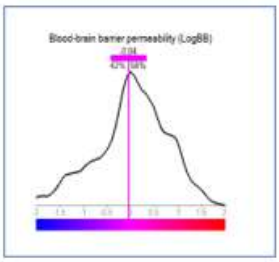
GLIPIZIDE DERIVATIVE I



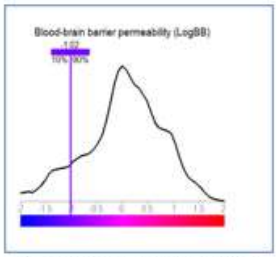
GLIPIZIDE DERIVATIVE II



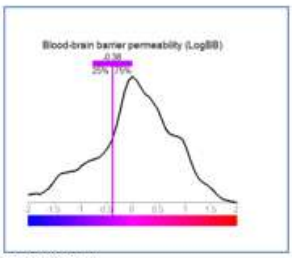
REPAGLINIDE



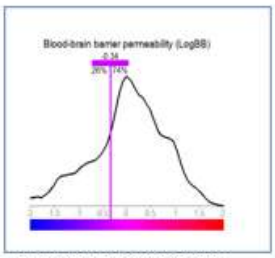
REPAGLINIDE DERIVATIVE I



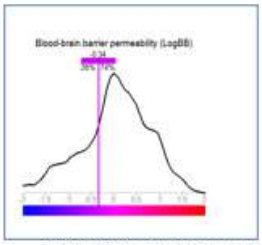
REPAGLINIDE DERIVATIVE II



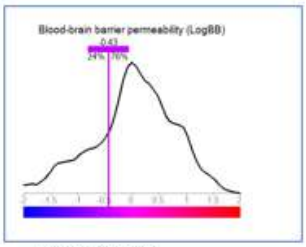
METFORMIN



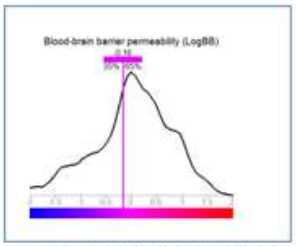
METFORMIN DERIVATIVE I



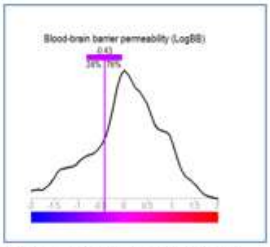
METFORMIN DERIVATIVE II



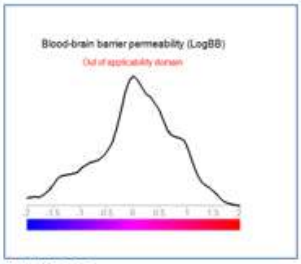
PIOGLITAZONE



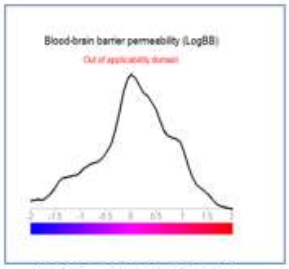
PIOGLITAZONE DERIVATIVE I



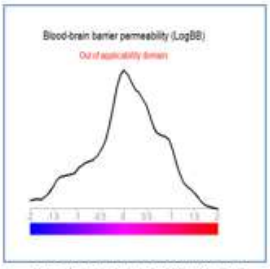
PIOGLITAZONE DERIVATIVE II



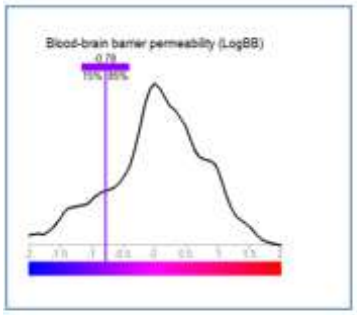
ACARBOSE



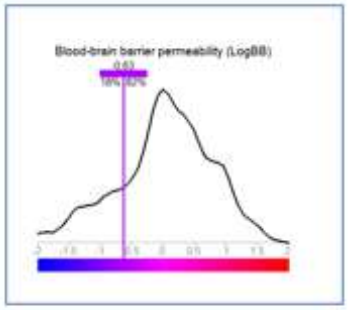
ACARBOSE DERIVATIVE I



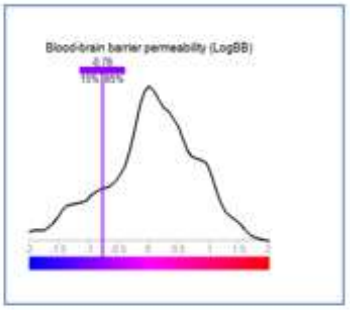
ACARBOSE DERIVATIVE II



DAPAGLIFLOZIN



DAPAGLIFLOZIN DERIVATIVE I



DAPAGLIFLOZIN DERIVATIVE II

PASS PREDICTION:

Activity	Chlorpropamide		Chlorpropamide d-I		Chlorpropamide d-II	
	Pa	Pi	Pa	Pi	Pa	Pi
Polyporopepsin inhibitor	0.856	0.0011	0.824	0.016	0.743	0.030
Omptin inhibitor	0.823	0.005	0.763	0.012	0.725	0.017
Cl-transporting ATPase inhibitor	0.766	0.009	0.805	0.005	0.740	0.011
Polyporopepsin inhibitor	0.856	0.0011	0.824	0.016	0.743	0.030

Activity	Glipizide		Activity		Glipizide	
	Pa	Pi	Pa	Pi	Pa	Pi
Sulfonylureas	0.674	0.001	0.421	0.002	0.673	0.001
Channel-conductance-controlling	0.520	0.010	0.518	0.010	0.510	0.011

ATPase inhibitor						
Potassium channel blocker	0.437	0.010	0.295	0.022	0.377	0.014
Prostaglandin E1 antagonist	0.370	0.010	0.374	0.010	0.351	0.012
Loop diuretics	0.357	0.042	0.351	0.044	0.336	0.053
Activity	**Repaglinide**		**Repaglinide d-I**		**Repaglinide d-II**	
	Pa	**Pi**	**Pa**	**Pi**	**Pa**	**Pi**
Insulin promoter	0.656	0.009	0.962	0.002	0.877	0.003
Antineurogenic pain	0.593	0.005	0.564	0.005	0.350	0.034
CYP3A4 inhibitor	0.443	0.008	0.461	0.007	0.417	0.010

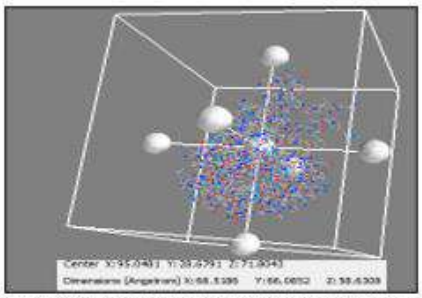
Activity	**Metformin**		**Metformin d-I**		**Metformin d-II**	
	Pa	**Pi**	**Pa**	**Pi**	**Pa**	**Pi**
CDP-glycerol glycerophosphotransferase inhibitor	0.887	0.012	0.898	0.010	0.848	0.020
Omptin inhibitor	0.820	0.006	0.837	0.005	0.735	0.015
Pro-opiomelanocortin converting enzyme inhibitor	0.824	0.011	0.844	0.009	0.785	0.016
Limulus clotting factor B inhibitor	0.808	0.004	0.827	0.004	0.713	0.011
Phobic disorders treatment	0.799	0.034	0.823	0.026	0.784	0.039

Activity	**Pioglitazone**		**Pioglitazone d-I**		**Pioglitazone d-II**	
	Pa	**Pi**	**Pa**	**Pi**	**Pa**	**Pi**
Antidiabetic	0.976	0.003	0.969	0.003	0.979	0.003
Peroxisome proliferator-activated receptor gamma agonist	0.942	0.002	0.920	0.002	0.944	0.002
CYP2C12 substrate	0.923	0.007	0.904	0.011	0.945	0.004
Peroxisome proliferator-activated receptor agonist	0.882	0.002	0.833	0.003	0.883	0.002
Hypolipemic	0.847	0.005	0.873	0.005	0.804	0.007
CYP2C8 substrate	0.820	0.005	0.877	0.004	0.882	0.004

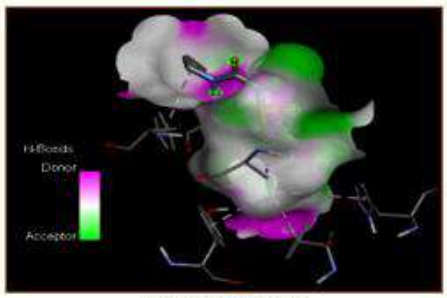
Activity	**Acarbose**		**Acarbose d-I**		**Acarbose d-II**	
	Pa	**Pi**	**Pa**	**Pi**	**Pa**	**Pi**

Cyclomaltodextrinase inhibitor	0.980	0.000	0.933	0.001	0.969	0.000
Sucrose alpha-glicosidase inhibitor	0.972	0.000	0.914	0.000	0.946	0.000
Maltose transporting ATPase inhibitor	0.968	0.000	0.916	0.000	0.928	0.000
4-Alpha glucanotransferase inhibitor	0.962	0.000	0.942	0.000	0.905	0.000
Alpha glucosidase inhibitor	0.956	0.000	0.933	0.000	0.910	0.000
CDP-glycerol glycerophosphotransferase inhibitor	0.949	0.004	0.940	0.004	0.938	0.005
Alpha amylase inhibitor	0.943	0.000	0.868	0.001	0.902	0.000
Glucan 1,3-alpha-glucosidase inhibitor	0.909	0.000	0.871	0.001	0.902	0.001

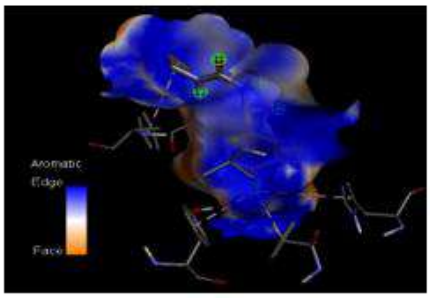
Activity	**Dapagliflozin**		**Dapagliflozin d-I**		**Dapagliflozin d-II**	
	Pa	**Pi**	**Pa**	**Pi**	**Pa**	**Pi**
Membrane integrity agonist	0.922	0.006	0.909	0.009	0.916	0.007
Antidiabetic	0.866	0.004	0.869	0.004	0.851	0.004
Alkenylglycerylphosphocholine hydrolase inhibitor	0.832	0.013	0.716	0.029	0.705	0.031
CDP-glycerol glycerophosphotransferase inhibitor	0.802	0.029	0.821	0.025	0.826	0.24
Sugar phosphate inhibitor	0.787	0.018	0.817	0.014	0.732	0.027
Cholesterol antagonist	0.754	0.005	0.672	0.011	0.670	0.011
Benzoate-CoA ligase inhibitor	0.749	0.024	0.765	0.022	0.721	0.028



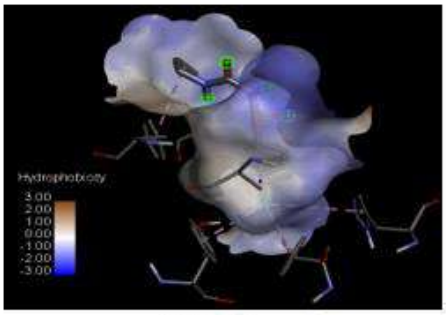
CHLORPROPAMIDE GRID BOX WITH LIGAND RECEPTOR



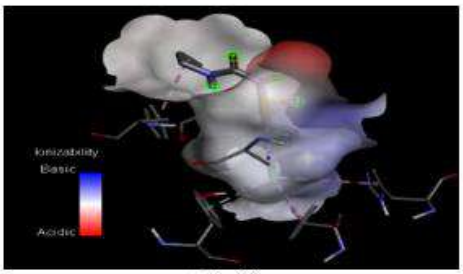
HYDROGEN BONDS



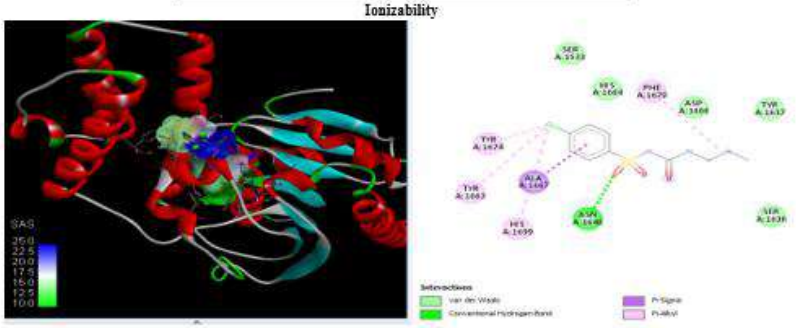
Aromatic



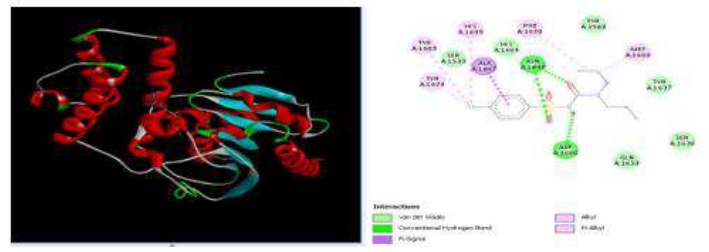
Hydrophobic



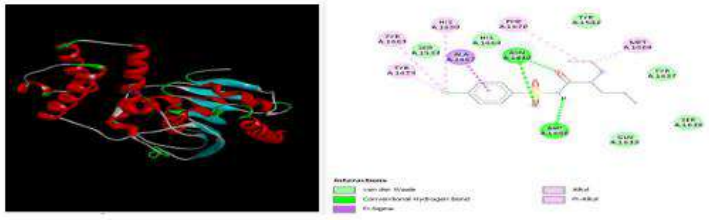
Ionizability



CHLORPROPAMIDE



CHLORPROPAMIDE I



CHLORPROPAMIDE II

Docking score of anti-tubercular protein polypeptide synthase pks13 (pdb id: 8q0t) and glycogen phosphorylase (pdb id: 1h5u)

Sr. No	Derivative Name	ANTI-TUBERCULAR PROTEIN POLYKETIDE SYNTHASE PKS13 (PDB ID: 8Q0T)Score	ANTI-DIABETIC PROTEIN GLYCOGEN PHOSPHORYLASE (PDB ID: 1H5U) Score
1	Chlorpropamide	-7.1	-10.9
2	Chlorpropamide I	-7.5	-7.2
3	Chlorpropamide II	-6.6	-8.1
4	Glipizide	-9.6	-4.6
5	Glipizide I	-9.5	-9.3
6	Glipizide II	-10.2	-8.8
7	Repaglinide	-9.8	-11.5
8	Repaglinide I	-9.6	-15.4
9	Repaglinide II	-9.0	-11.4
10	Metformin	-11.2	-9.4
11	Metformin I	-10.5	-4.8
12	Metformin II	-10.8	-4.3
13	Pioglitazone	-7.5	-12.3
14	Pioglitazone I	-7.7	-8.0
15	Pioglitazone II	-7.4	-8.1
16	Acarbose	-8.8	-7.2
17	Acarbose I	-8.5	-11.5
18	Acarbose II	-8.4	-13.1
19	Dapagliflozin	-10.2	-11.3
20	Dapagliflozin I	-10.3	-10.3
21	Dapagliflozin II	-10.0	-9.1

CONCLUSION:

Successfully demonstrated the potential of novel derivatives of standard antidiabetic drugs, exhibiting reduced toxicity and enhanced efficacy through in-silico ADME/T studies and molecular docking. The significant interactions with

antidiabetic and antitubercular properties, confirmed by docking with protein PDB IDs 8Q0T and 1H5U, and predictive Pa and Pi values (0-1), underscore the promise of these derivatives as next-generation antidiabetic compounds. This study provides valuable leads for future research, paving the way for the development of more effective and safer antidiabetic therapies, and highlighting the potential of computational drug design in accelerating the drug discovery process.

ACKNOWLEDGEMENT

My greatest thanks goes out to the principal and professors of the SSM College of Pharmacy and study Center at BLDEA's in Vijayapura for their steadfast encouragement and moral support during my research work.

CONFLICT OF INTEREST:

No

REFERENCES:

1. Arif R, Ahmad S, Mustafa G, Mahrosh HS, Ali M, Tahir ul Qamar M, Dar HR. Molecular Docking and Simulation Studies of Antidiabetic Agents Devised from Hypoglycemic Polypeptide-P of Momordica charantia. BioMed research is international. 2021;2021(1):5561129.
2. Zabidi NA, Ishak NA, Hamid M, Ashari SE, Mohammad Latif MA. Inhibitory evaluation of Curculigo latifolia on α-glucosidase, DPP (IV) and in vitro studies in antidiabetic with molecular docking relevance to type 2 diabetes mellitus. Journal of Enzyme Inhibition and Medicinal Chemistry. 2021 Jan 1;36(1):109-21.
3. Chinthala Y, Thakur S, Tirunagari S, Chinde S, Domatti AK, Arigari NK, Srinivas KV, Alam S, Jonnala KK, Khan F, Tiwari A. Synthesis, docking and ADMET studies of novel chalcone triazoles for anti-cancer and anti-diabetic activity. European journal of medicinal Hussain N, Kakoti BB, Rudrapal M, Sarwa KK, Celik I, Attah EI,
4. Addanki HR, Vallabhaneni MR, Chennamsetty S, Pullagura P, Sagurthi SR, Pasupuleti VR. An in silico ADMET, molecular docking study and microwave-assisted synthesis of new phosphorylated derivatives of thiazolidinedione as potential anti-diabetic agents. Synthetic Communications. 2022 Jan 17;52(2):300-15.
5. Maji D, Samanta S, Patil VM. In silico ADMET and docking studies of thiazolidinedione-acetic-acid hybrids as antidiabetics with cardioprotection. Letters in Drug Design & Discovery. 2020 Dec 1;17(12):1475-84.
6. Salgueiro AC, Folmer V, Bassante FE, Cardoso MH, da Rosa HS, Puntel GO. Predictive antidiabetic activities of plants used by persons with Diabetes mellitus. Complementary Therapies in Medicine. 2018 Dec 1; 41:1-9.
7. Dash L, Sharma N, Vyas M, Kumar R, Kumar R, Mahajan S, Khurana N. Prediction of anticancer activity of potential anticancer compounds using pass online software. Plant Arch. 2020;20(2):2808-13.
8. Akash S. Computational screening of novel therapeutic and potent molecules from bioactive trehalose and it's eight derivatives by different insilico studies for the treatment of diabetes mellitus. Organic Commun. 2022 Jul 1; 15:288-96.

HOW TO CITE: Anushree Suga , Ashwini Paschapur , Somashekhar M. Metri , Trupti A. Hunnura , Hanamant B. Sannakki , An Approach Of Computer Aided Drug Design (CADD) Tools For In-silico Evaluation Of Various Derivatives Of Antidiabetic Standard Drugs, Int. J. of Pharm. Sci., 2024, Vol 2, Issue 10, 987-998. https://doi.org/10.5281/zenodo.13955358