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Molecular Docking of Some Novel Quinoline Derivatives as Potent Inhibitors of Human Breast Cancer Cell Line

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ABSTRACT. Breast Cancer is one of the major universal health problems affecting more than one million cases per year. Incidence of breast cancer would be seriously increased by inefficacy of the existing available drugs; therefore, designing novel drugs is almost a crucial issue for medication of breast cancer. In this work, some novel synthesized derivatives of quinoline were examined against human breast cancer cell line (MCF-7) through performing structural optimizations and molecular docking simulations to evaluate the binding affinity against topoisomerase (ii) (Topo2α) receptor target. Indeed, first-hand information for the design of novel and potent drugs for medication of breast cancer compounds were provided here. Molecular docking processes were carried out with the help of AutoDock-Vina of PyRx and Discovery Studio software programs. Evaluated binding scores indicated that ligand number 29 could work properly with the lowest binding energy value of -10.4 kcal/mol among 31 investigated ligands. Furthermore, this ligand showed higher binding affinity and bonding strength to the pocket of receptor target (Topo2α) in comparison with the hypothetical Doxorubicin reference drug with binding energy of -6.9 kcal/mol. The provided results of this work could be useful for those researchers working on designing novel medication protocols for breast cancer specially based on quinoline derivatives.

KEYWORDS. Breast cancer; Binding affinity; Topoisomerase (ii); Doxorubicin; Quinoline; Docking.

INTRODUCTION. Cancer is an abnormal growth of the cells leading to one of the most critical health issues for humankind worldwide with deathful effects. Despite availability of the improved drugs for targeted cancer therapies, but huge numbers of cancer panties and deaths every year shows inefficiency of current medication protocols.[1] Breast cancer has been seen as an epidemic posing a serious threat to the health of women of all races globally, in which about numbers of new cases are arising ever year all around the world.[2] In Nigeria, cervical cancer was the commonest cause of cancer related deaths among women for decades, breast cancer is now the leading cause of most cancer related deaths and this is not due to the reduction in cervical cancer but an increase in the incidence of breast cancer.[3] Current therapeutic treatments of cancer are usually focused on targeting critical cellular processes involved in DNA replication and cell division.

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This method consists of different sets of agents of each targeting different pathways and enzymes. Among which, drug targeting DNA topoisomerases has been seen as predominantly effective at disrupting cancer cell growth.[4] They are a family of enzymes originated from the nucleus and the mitochondria, which are responsible for maintaining DNA topology.[5] DNA topology refers to relationship of two strands of the double helix and it includes the concept of supercoiling.[6] Type II topoisomerases (Topo2α) forms a transient double strand DNA break in one segment passing one DNA segment to another through the break prior to ligating the cleaved DNA ends. Topo2α is divided into IIA and IIB varying in terms of structure, mechanism and cofactor found in living organisms.[7]. These enzymes could work either to enhance different chromosomes e.g., for chromosome segregation and unknotting, or sections of the same chromosome e.g., during transcription and replication.[8]

Molecular docking is a computational technique for predicting accurate values of binding scores ligand-target interacting complexes.[9] The derived information could be used to evaluate the energy profiling, such as binding energy, interacting bond length, strength and binding constant. Current use of molecular docking has been mainly aimed to calculate interaction strengths and quality between the micromolecular ligands and macromolecular protein targets in order to define their tentative parameters.[10] The calculated binding parameters would then serve as raw data for rational drug design of structure based drug development (SBDD) of new agents with better efficacy.[11] Using such benefit, potency of some novel quinoline derivatives for inhibiting breast cancer were investigated in this computer-based work.

METHODOLOGY. Molecular docking processes of this work were carried out on 31 quinoline derivatives against Topo2α receptor target to elucidate the binding mode of ligand-target complexes. These ligand compounds with reported inhibitory activity against human breast cancer cell line (MCF-7) were retrieved from the literature. 2D structure of the compounds were drawn with ChemDraw software and they were imported into the Spartan software to obtain the optimized 3D spatial conformers. The optimized 3D structures were then converted to protein data bank format (PDB) to be included in the material studio software.[12] Content of Table 1 presented the structures and activities of the ligand compounds. 3D structure of Top2α was retrieved from RCSB with PDB code of 4fm9.[13] The receptor target was prepared by removing all the attached substance such as cofactors, water molecules and already included ligands (Fig. 1).[14] The ligands were also prepared by simply converting PDB format of the optimized 3D structures from Spartan to each of pdbq and pdbqt formats for inclusion in molecular docking processes.[15] The prepared receptor target was imported into the PyRx virtual screening tool and saved as macromolecule, the ligands were imported one after the other into the same tool. Molecular docking processes were performed for the selected items by running AutoGrid and AutoDock commands tools using the AutoDock-Vina of PyRx software. Values of binding energy of the interacting ligand-target complexes were calculated and the obtained complexes were visually analyzed by the Discovery Studio software program. It is important to note that such processes have been seen as the standard methodology for analyzing drug-receptor interactions as an advantage of computer-based works for investigating biological related systems.[16-20] All obtained content of this work were summarized in Tables 1 and 2 and Figs. 1-3 for further discussing about the proposed problem.

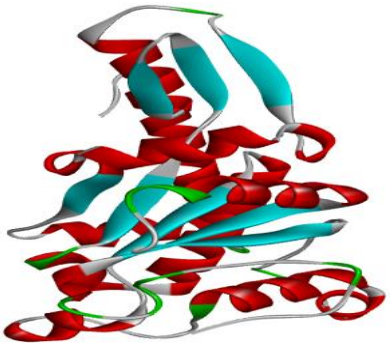


Fig. 1: 3D representation of Topo2α.

RESULTS & DISCUSSION. Within this work, molecular docking processes of 31 quinoline derivatives (Table 1) were investigated towards the 3D structure of Top2α target (Fig. 1). All the materials were prepared for providing required results for discussing on design of novel inhibitors for breast cancer problem. The obtained results were all summarized in Table 2 and Figs. 2 and 3 for the models.

Table 1. Ligands 1-16 representations.

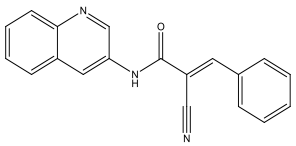
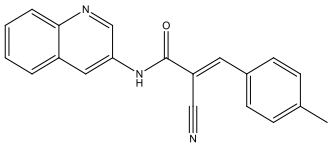
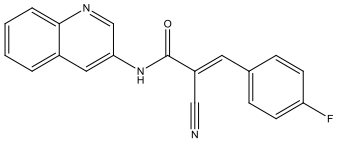
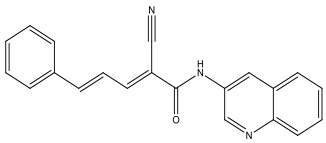
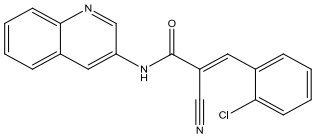
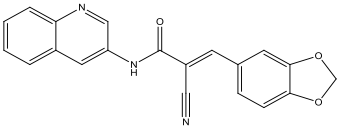
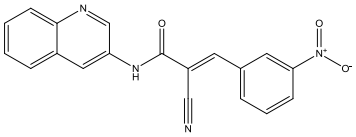
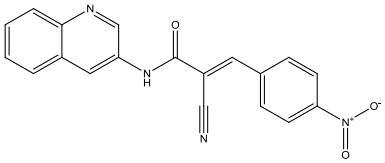
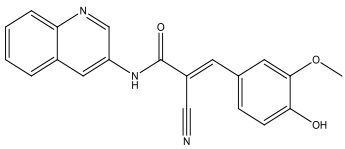
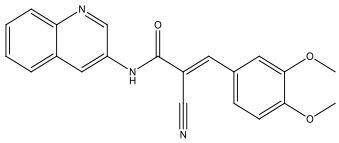
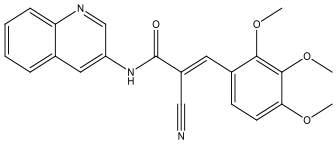
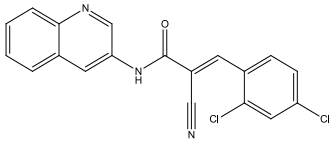
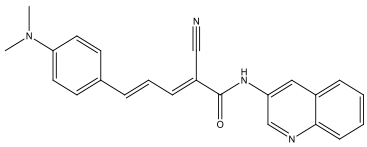
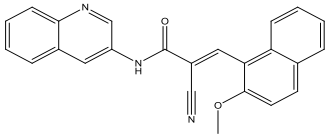
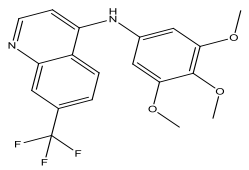
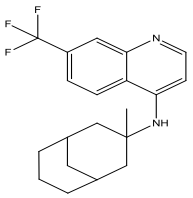
No.	Structure and IUPAC Name	IC_{50}	PIC_{50}
1	 2-cyano-3-phenyl-N-(quinolin-3-yl)acrylamide	79.20	4.10
2	 2-cyano-N-(quinolin-3-yl)-3-p-tolylacrylamide	74.40	4.13
3	 2-cyano-3-(4-fluorophenyl-N-(quinolin-3-yl)acrylamide	40.00	4.40
4	 2-cyano-5-phenyl- N-(quinolin-3-yl) penta-2,4-dienamide	63.60	4.20
5	 3-(2-chlorophenyl)-2-cyano-N-(quinolin-3-yl) acrylamide	53.50	4.27
6	 3-(benzo[1,3]dioxol-5-yl)-2cyano-N-(quinolin-3-yl)acrylamide	57.10	4.24
7	 2-cyano-3-(3-nitrophenyl)-N-(quinolin-3-yl)acrylamide	65.20	4.19
8	 2-cyano-3-(4-nitrophenyl)-N-(quinolin-3-yl)acrylamide	63.00	4.20
9	 2-cyano-3-(4-hydroxy-3-methoxyphenyl)-N-(quinolin-3-yl)acrylamide	29.80	4.52
10	 2-cyano-3-(3,4-dimethoxyphenyl)-N-(quinolin-3yl) acrylamide	64.60	4.19
11	 2-cyano-N-(quinolin-3-yl)-3-(2,3,4-trimethoxyphenyl)acrylamide	49.80	4.30
12	 2-cyano-3-(2,4-dichorophenyl)-N-(quinolin-3-yl) acrylamide	57.60	4.24
13	 2-cyano-5-(4-(dimethyl amino) phenyl)-N-(quinolin-3-yl) penta-2,4-dienamide	40.40	4.39
14	 2-cyano-3-(2methoxynaphthalen-1-yl)-N-(quinolin-3-yl) acrylamide	57.50	4.24
15	 7-(trifluoromethyl)-N-(3,4,5-trimethoxyphenyl) quinolin-4-amine	9.38	5.03
16	 N-(3-methyl bicyclo[3.3.1]nonan-3-yl) 7(trifluoromethyl)quinolin-4-amine	24.10	4.62

Table 1 (continued). Ligands 17-31 representations.

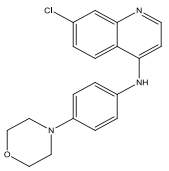
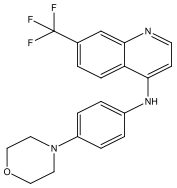
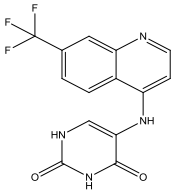
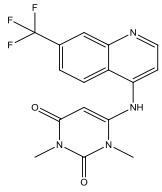
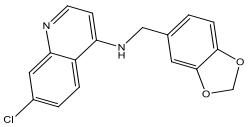
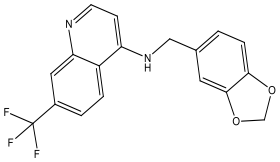
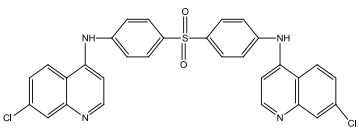
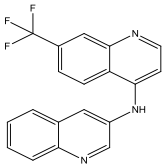
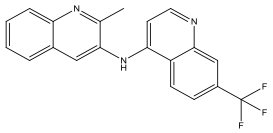
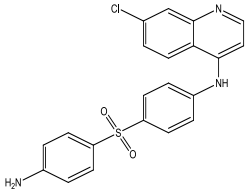
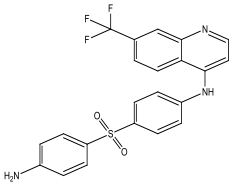
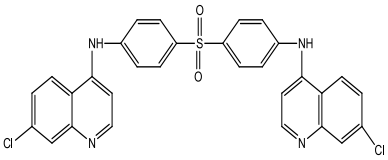
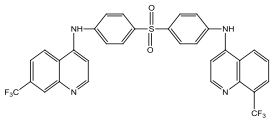
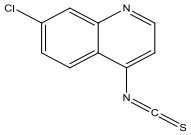
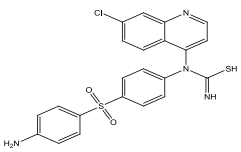
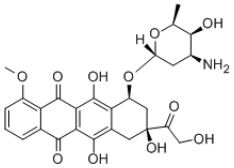
No.	Structure and IUPAC Name	IC_{50}	PIC_{50}
17	 7-chloro-N-(4-morpholinophenyl) quinolin-4-amine	31.50	4.50
18	 N-(4-morpholinophenyl)-7-(trifluoromethyl) quinolin-4amine	23.30	4.63
19	 5-(7-(trifluoromethyl) quinolin-4-ylamino) pyrimidin-2,4 (1H,3H)-dione	21.40	4.67
20	 1,3-dimethyl-6-(7-(trifluoromethyl) quinolin-4-ylamino) pyrimidin-2,4-(1H,3H)-dione	23.30	4.63
21	 N-(benzo[d] [1,3] dioxol-5-ylmethyl)-7-chloroquinolin-4-amine	21.10	4.68
22	 N-(benzo[d] [1,3] dioxol-5-ylmethyl)-7-(trifluoromethyl)-quinolin-4-amine	26.20	4.58
23	 N-(5,6-dimethyl-1,2,4-triazin-3-yl)-7-(trifluoromethyl)-quinolin-4-amine	21.80	4.66
24	 N-(7-(trifluoromethyl)-quinolin-4-yl)-quinolin-3-amine	14.20	4.85
25	 2-methyl-N-(7-trifluoromethyl) quinolin-4-yl)-quinolin-3-amine	16.30	4.79
26	 N-(4-(4-aminophenylsulfonyl) phenyl)-7-chloroquinolin-4-amine	18.80	4.72
27	 N-(4-(4-aminophenylsulfonyl) phenyl-7-(trifluoromethyl)-quinolin-4-amine	23.50	4.63
28	 N,N'-(4,4'-sulfonylbis(4,1-phenylene)bis(7-chloroquinolin-4-amine)	23.20	4.63
29	 N, N'-(4,4'sulfonylbis(4,1-phenylene) bis(7-(trifluoromethyl)-quinolin-4-amine)	24.00	4.62
30	 7-Chloro-4-isothiocyanatoquinoline	22.40	4.65
31	 N-(4-(4-aminophenylsulfonyl) phenyl)-N-(7-chloroquinolin-4-yl)-carbamimiodothioic acid	22.70	4.64
REF	 Doxorubicin	n/a	n/a

Table 2. Molecular docking specifications of complexes 1-16.

No.	Binding Energy	Hydrogen Bond Interactions		Hydrophobic Interactions
	kcal/mol	Amino Acid	Length Å	Amino Acid
1	-8.2	SER547	2.60	ALA652, PHE653, LEU565, ILE554, LYS639,
2	-8.3	n/a	n/a	LEU565, ILE554, PHE653, ALA652
3	-8.5	SER547	2.35	ALA652, ILE554,LEU565, PHE653
4	-9.0	ALA588	2.14	HIS634
		ARG633	4.59	
5	-8.2	HIS567	2.17	LEU528
		ARG568	3.59	
6	-9.0	ALA588	2.81	GLU626, HIS634, GLU586, PHE589
		ARG633	2.00	
		GLN594	2.75	
7	-8.2	ARG635	6.07, 3.07	LEU528, PHE569
		HIS597	2.25	
8	-8.3	ALA588	2.46	ALA629
		HIS634	2.31	
		TYR590	2.71	
9	-8.0	SER547	2.13	ALA625, ILE554, PHE653
10	-8.1	HIS567	2.26	LEU528
11	-7.3	THR453	3.02	LEU528
		LYS535	2.42	
		HIS567	2.26	
12	-8.3	ASP645	2.86	ALA648, LEU565, LEU570, ILE554, PHE638
13	-8.7	HIS548	2.96	ILE554, ILE665, ARG661
14	-8.9	TYR590	2.5	ASP630, GLU586, PHE
		ARG633	2.23	
		GLU594	2.57	
		ALA588	2.55	
15	-7.6	LYS639	2.80	ALA648, ILE554, LEU565, LEU570, PHE638
		ASP645	2.89	
16	-8.9	HIS567	2.22	LEU528, GLY534,TYR533, LEU531, PHE569
		THR453	2.42	

Molecular docking specifications for ligand-target interacting complexes of quinoline derivatives and Top2α target were all summarized in Table 2. It is here important to mention that both of quantities and qualities are important for describing interacting complexes, in which values of binding energy and lengths could describe quantity side and types of interactions and amino acids could describe quality side of such ligand-target complex analyses. In this work, all required parameters were provided to achieve the purpose of designing novel quinoline based inhibitors for breast cancer target receptor. The results indicated that almost all ligands were in strong interaction with the target. Their binding energy values were ranged from -5.8 to -10.4 kcal/mol for complex formations. Ligand number 29 was seen with the most favorable value of binding energy of -10.4 kcal/mol meaning that the complex formation of this ligand and Top2α target was the strongest one among other complexes of such involving systems. To show the importance of such strength, the value was compared with the binding energy of Doxorubicin as a reference drug for interacting with the same target. The value of binding energy of Doxorubicin-Top2α complex formation was -6.8 kcal/mol significantly lower in strength than that of value obtained for ligand number 29. Here it is important to mention that careful modification of the chemical structures could yield to design new inhibitors with more potency and efficiency towards the targets in biological media. Indeed, such chemical modifications could be done better regarding the importance of lead compounds or reference compounds to be proposed more properly for further investigations of drug design. For the case of cancer with so many types of complexity for performing systematic investigations *in vitro* or *in vivo*, performing such *in silico* works could provide insightful information in the lowest molecular scales.[21, 22] Indeed, this is an advantage in both of predictions and interpretations of the experimental achievements.

Table 2 (continued). Molecular docking specifications of complexes 17-31.

No.	Binding Energy	Hydrogen Bond Interactions		Hydrophobic Interactions
	kcal/mol	Amino Acid	Length Å	Amino Acid
17	-8.1	LYS639	2.21	ALA652, ALA648, ILE649, ILE554, LEU570, EU565, PHE638, PHE653
18	-8.7	LYS639	2.96	SER547, PHE653, ALA652, ALA648, ILE649,
		ASP645	2.85	ILE554, PHE638
19	-7.8	GLU586	4.83	ALA588, ALA629
		GLU626	2.23	
		ARG633	2.82	
		TYR590	2.36	
20	-8.2	THR453	2.69	GLU454, GLU525, LEU528, LEU564, PHE569
		HIS567	2.10	
21	-7.7	THR453	2.60	LEU531, LEU528
		HIS567	2.87	
		ARG568	2.72	
22	-7.9	LYS639	2.87	ALA652, ALA648, ILE649, ILE554, LEU565,
		ASP645	2.72	LEU570, PHE638
23	-9.8	ASP645	2.46	ALA647, ALA648, ILE554, LEU565, LEU570,
		ASP660	2.99	LEU651
		TRP664	3.08	
		LYS639	2.82	
24	-8.7	HIS567	2.08	GLY534, TRY533, LEU531, LEU528
		THR453	2.48	
25	-9.1	ASP645	2.32	ALA652, ILE554, ILE649, LEU565, LYS639
26	-8.3	GLY551	3.01	ALA652, ASP645, ILE554, LEU565, LEU570
		LYS639	1.79	
27	-8.8	THR453	2.32	LEU528, LEU531, TYR533, GLY534
		HIS567	2.94	
		ASP524	2.34	
28	-9.3	LYS639	1.81	ASP645, ALA652, ARG661, ILE554, LEU565,
		GLY551	3.01	LEU570, LEU665
29	-10.4	LYS639	2.33	ASP645, ALA648, ALA652, LYS550, ILE574,
		GLN542	2.93	LEU565, GLY551, GLU572, PHE638
30	-5.8	THR453	2.05	LEU528, LEU531, LEU564, PHE569
		HIS567	1.89	
31	-7.6	GLY551	2.84	ASP645, ALA652, LEU565,LEU570, ILE554,
		LYS639	2.30	SER547, PHE638
REF	-6.8	LEU516	2.39	GLN517, ARG532
		ASN433	2.00	
		THR530	2.95	
		LYS520	2.95	

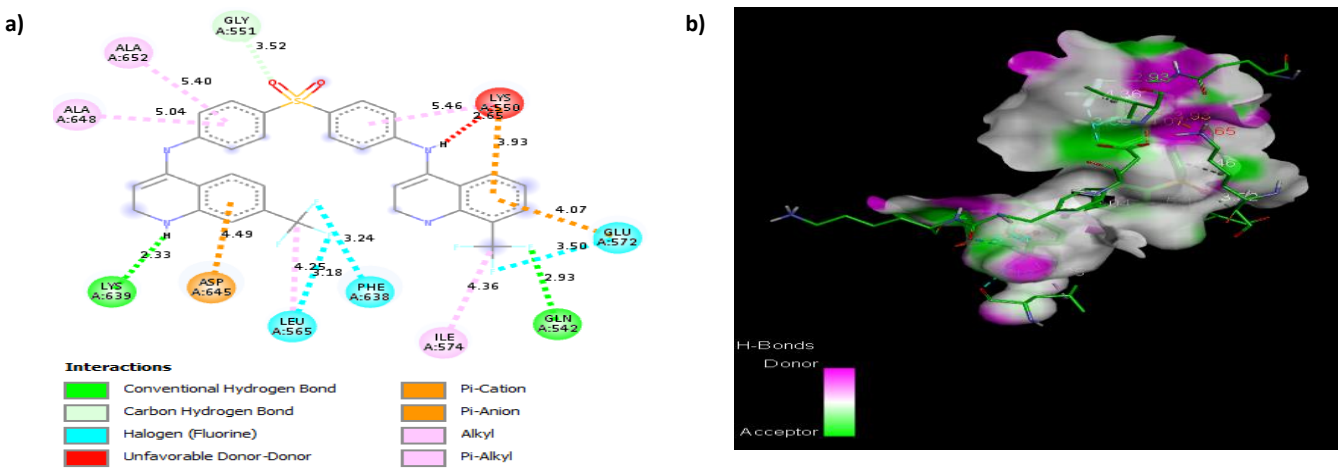


Fig. 2: a) 2D and b) 3D representations of complex of ligand number 29 and Topo2α target.

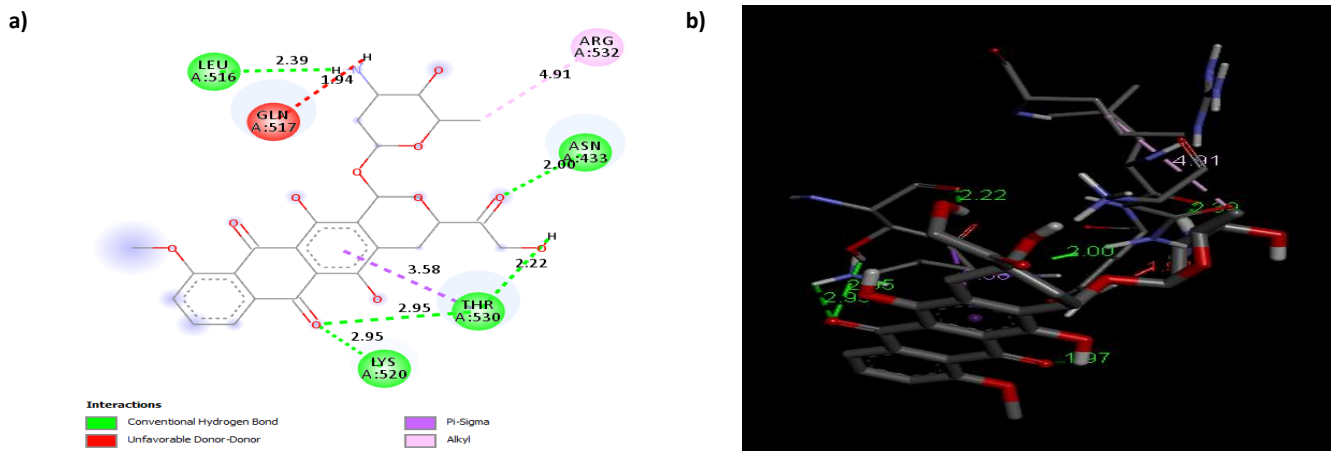


Fig. 3: a) 2D and b) 3D representations of complex of reference Doxorubicin and Topo2α target.

The hydrogen bond and hydrophobic interactions of complex formations between the ligands and target were completely described in Table 2 and those of ligand number 29 and reference Doxorubicin were exhibited in Figs. 2 and 3. As described in Table 2, ligand 29 was introduced as the optimum one among the ligands with two hydrogen bond interactions of 2.33 and 2.93 Å with LYS639 and GLN542 of the target respectively. Furthermore, many hydrophobic interactions with ASP645, ALA648, ALA652, LYS550, ILE574, LEU565, GLY551, GLU572, and PHE638 of the target site were indicated as shown in Fig. 2. The H-F group in 8-fluoro-4-methyl-1,2-dihydroquinoline act as donor and form a hydrogen bond with GLN542 residue whereas the N-H group in 1,2-dihydroquinoline-4-amine act as an acceptor and form a hydrogen bond also with LYS639 of the target. The reference Doxorubicin drug was also docked with the topo2α target to show evidences of applicability of investigated ligands for the purpose of cancer growth inhibition. As shown described in Table 2 and shown Fig. 3, it was found that four conventional hydrogen bond interactions with lengths of 2.00, 2.39, 2.95, and 2.95 Å were found with amino acids of ASN433, LEU516, LYS20 and THR530 of the target. It was also found that two hydrophobic interactions with GLN517 and ARG532 were found for Doxorubicin-topo2α complex system. The amine (-NH_2) group in 2,3,6-trimethyltetrahydro-2H-pyran-4-amine of Doxorubicin acted as a donor and formed a hydrogen bond interaction with LEU516, the carbonyl group (-C=O) in 2-methoxyanthracene-9,10-dione also acted as donor and formed two hydrogen bond interactions with THR530 and LYS520. However, the hydroxyl group (-OH) group and the carbonyl group (-C=O) in 2-hydroxyacetaldehyde of Doxorubicin acted as acceptors and formed hydrogen bond interactions with THR530 and ASN433 respectively.

CONCLUSION. In conclusion, the main aim of this *in silico* based research was achieved successfully by obtained features of molecular docking processes. All investigated ligands showed favorable features for interactions with the target, in which ligand number 29 was seen the most favorable one among the ligands and also reference Doxorubicin. Significant value of binding energy of -10.4 kcal/mol introduced ligand number 29 as a proper ligand for running further examinations on it. As a consequence, this study showed the advantage of performing *in silico* work to serve quinoline derivatives for possible inhibitions of breast cancer growth avoiding harmful and dreadful effects for those related patients.

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REFERENCES

1. Frankish H. 15 million new cancer cases per year by 2020, says WHO. Lancet. 2003;36:1278-1287.
2. Cancer facts & figures. American Cancer Society. The Society. 2008.
3. Okobia MN, Bunker CH, Okonofua FE, Osime U. Knowledge, attitude and practice of Nigerian women towards breast cancer: a cross-sectional study. World Journal of Surgical Oncology. 2006;4:11-15.

4. Nitiss JL. DNA topoisomerase II and its growing repertoire of biological functions. Nature Reviews Cancer. 2009;9,327-337.
5. Pommier Y, Sun Y, Shar-yin NH, Nitiss JL. Roles of eukaryotic topoisomerases in transcription, replication and genomic stability. Nature Reviews Molecular Cell Biology. 2016;17:703-721.
6. Deweese JE, Osheroff MA, Osheroff N. DNA topology and topoisomerases: teaching a "Knotty" subject. Biochemistry and Molecular Biology Education. 2009;37:2-10.
7. Forterre P, Gadelle D. Phylogenomics of DNA topoisomerases: their origin and putative roles in the emergence of modern organisms. Nucleic Acids Research. 2009;37:679-692.
8. Pommier Y, Sun Y, Huang SN, Nitiss JL. Roles of Eukaryotic Topoisomerases in transcription, replication and genomic stability. Nature Reviews Molecular Cell Biology. 2016;17:703-721.
9. Mukesh B, Rakesh K. Molecular docking: a review. International Journal of Research in Ayurveda and Pharmacy. 2011;2:1746-1751.
10. Farahbakhsh Z, Zamani MR, Rafienia M, Gülseren O, Mirzaei M. In silico activity of AS1411 aptamer against nucleolin of cancer cells. Iranian Journal of Blood and Cancer. 2020;12:95-100.
11. Guedes IA, de Magalhães CS, Dardenne LE. Receptor-ligand molecular docking. Biophysical Reviews. 2014;6:75-87.
12. Rose PW, Beran B, Bi C, Bluhm WF, Dimitropoulos D, Goodsell DS, Prlić A, Quesada M, Quinn GB, Westbrook JD, Young J. The RCSB protein data bank: redesigned web site and web services. Nucleic Acids Research. 2010;39:392-401.
13. Mirali M, Jafariazar Z, Mirzaei M. Loading tacrine Alzheimer's drug at the carbon nanotube: DFT approach. Lab-in-Silico. 2021 3;2:3-8.
14. Khalid H, Hussain R, Hafeez A. Virtual screening of piperidine based small molecules against COVID-19. Lab-in-Silico. 2020;1:50-55.
15. Mirzaei M, Harismah K, Da'I M, Salarrezaei E, Roshandel Z. Screening efficacy of available HIV protease inhibitors on COVID-19 protease. Journal Military Medicine. 2020;22:100-107.
16. Harismah K, Mirzaei M. Steviol and iso-steviol vs. cyclooxygenase enzymes: in silico approach. Lab-in-Silico. 2020;6:11-15.
17. Dizdaroglu Y, Albay C, Arslan T, Ece A, Turkoglu EA, Efe A, Senturk M, Supuran CT, Ekinci D. Design, synthesis and molecular modelling studies of some pyrazole derivatives as carbonic anhydrase inhibitors. Journal of Enzyme Inhibition and Medicinal Chemistry. 2020;35:289-297.
18. Yamali C, Gul HI, Ece A, Bua S, Angeli A, Sakagami H, Sahin E, Supuran CT. Synthesis, biological evaluation and in silico modelling studies of 1, 3, 5-trisubstituted pyrazoles carrying benzenesulfonamide as potential anticancer agents and selective cancer-associated hCA IX isoenzyme inhibitors. Bioorganic Chemistry. 2019;92:103222.
19. Mirzaei M, Hadipour NL. An investigation of hydrogen-bonding effects on the nitrogen and hydrogen electric field gradient and chemical shielding tensors in the 9-methyladenine real crystalline structure: a density functional theory study. The Journal of Physical Chemistry A. 2006;110:4833-4838.
20. Harismah K, Ozkendir OM, Mirzaei M. Lithium adsorption at the C20 fullerene-like cage: DFT approach. Advanced Journal of Science and Engineering. 2020;1:74-79.
21. Mirzaei M. Science and engineering in silico. Advanced Journal of Science and Engineering. 2020;1:1-2.
22. Mirzaei M. Making sense the ideas in silico. Lab-in-Silico. 2020;1:31-32.

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