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Mucuna pruriens (L.)- A Potential Phospholipase A_2 Inhibitor: *In silico* Approach

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Abstract: In this study, the function of phytochemicals in the active site of the examined target was explored, along with the prediction of potent ligands that exhibit significant biological activity through in silico methods. Thus, eight (8) phytochemicals located in *Mucuna pruriens* (L.) were retrieved from the PubChem database, and they were studied using Spartan 14 software, the molecular operating environment, AdmetSar software, and Gromacs. The analyses performed revealed the activities of the compounds under investigation. The molecular interactions between the ligands studied and Phospholipase A2 (PDB ID: 1gp7) indicated that bis(2-ethylhexyl) benzene-1,2-dicarboxylate (Compound 4) exhibited the highest inhibitory capacity against Phospholipase A2 when compared to the other compounds examined, as well as the reference compound. The binding affinity of compound 4 (-8.25364113 kcal/mol) was assessed against the binding affinity of the reference compound (-7.45687246 kcal/mol), demonstrating that compound 4 possesses greater inhibitory potential than the reference compound. Furthermore, the effectiveness of compound 4 was validated through molecular dynamics simulations, and the physicochemical properties of both the lead compound and the reference compound were evaluated using absorption, distribution, metabolism, excretion, and toxicity (ADMET) software. Our discoveries may open the door for the design of a collection of proficient bis(2-ethylhexyl) benzene-1,2-dicarboxylate-based drug-like compounds as potential anti-venom agents.

Keywords: *Mucuna pruriens*; phospholipase; *In silico*; snake; docking.

1. Introduction

Throughout history, snakes have been among the reptiles most feared and loathed by humans [1]. In certain Indian cultures, certain snakes, such as the cobra, are revered as sacred [2]. Furthermore, governments worldwide continue to strive to mitigate the impact of snake bite envenomation in tropical regions, yet their efforts seem endless. Reports indicate that many rural areas lack modern health services, and some have no healthcare facilities at all, resulting in the deaths of numerous rural residents [3-6]. A study by Theakston and Warrell in 2000 highlighted a shortage of antivenom in various parts of the world [7]. In Nigeria—a nation in West Africa—over 150 snakebite cases were reported for every 99 healthcare instances admitted to hospitals [8]. Despite the rising number of snakebite incidents in Africa and Asia, the production rate of anti-venom has been reported to decline, with some anti-venom products lacking efficacy against the venom's effects on the human body. Additionally, even the more effective products are often unaffordable for rural populations, leading to many fatalities [9].

Phospholipase A_2 has been categorized as a crucial enzyme that catalyzes the cleavage of fatty acids at the ortho-position of phospholipids. It also hydrolyzes the link between glycerol and the tail end of the second fatty acid [10]. This enzyme is usually located in the tissues of mammals. It can also be found in the tissues of snake venom and arachnid venom, among others. According to some researchers, melittin, which kindles Phospholipase A_2, could be found in bees' venom in large amounts, and Phospholipase A_2 has also been observed to be present in large amounts in snakes, which leads to uneven discharge of arachidonic acid from the phospholipid membrane, thereby leading to pain at the spot of attack [11-14]. Also, in snakes, Phospholipase A_2 (PLA_2) is a key component of venom, contributing to its toxicity and physiological effects. Snake venom PLA_2s can disrupt cell membranes by hydrolyzing phospholipids, leading to cell lysis, tissue damage, and inflammation. These enzymes also play roles in neurotoxicity, myotoxicity, anticoagulation, and edema, depending on the snake species. Their diverse toxic effects aid in immobilizing prey, initiating digestion, and defending against predators.

Thus, researching medicinal plants with the potential to inhibit Phospholipase A2, thereby reducing the havoc caused by snakes in rural areas, would be a significant advantage.

Mucuna pruriens is classified as a member of the Fabaceae family and is commonly found in tropical regions [15]. Moreover, it is commonly recognized for its itching characteristics, and according to Emenalon *et al.*, it can be consumed as food by humans [16]. Kamkaen *et al.* also stated that the seeds of Mucuna pruriens are utilized as a nerve tonic and serve as an enhancement for men [17]. Hence, this study aims to investigate the role of phytochemicals in the active site of the receptor under analysis, as well as to design effective compounds with improved anti-Phospholipase A_2 properties using in silico methods.

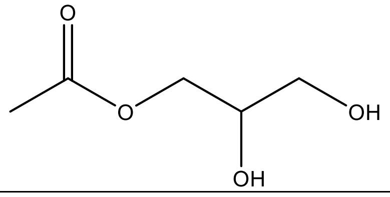
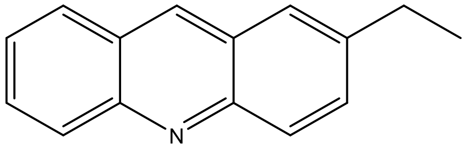
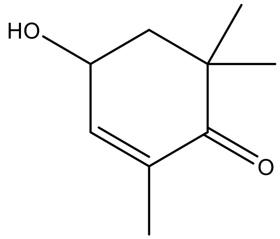
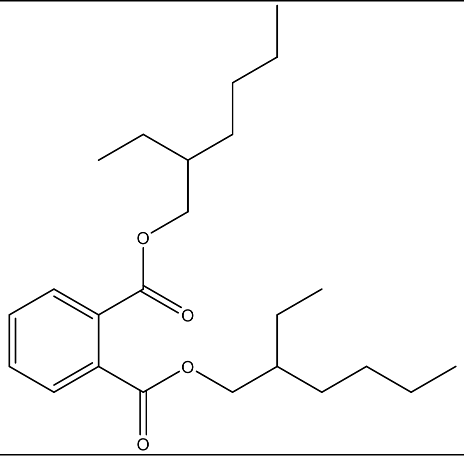
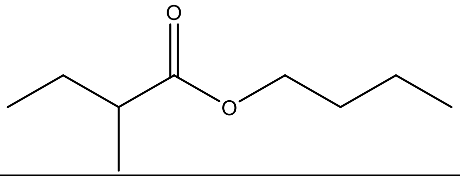
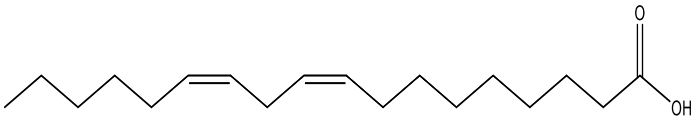
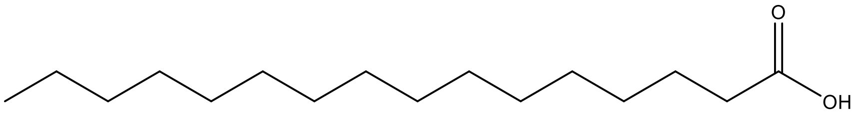
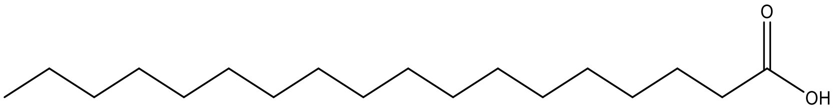
2. Materials and Methods

2.1. Density functional theory-based optimization.

The selected compounds [18] used in this work were optimized using Spartan 14 software with a 6-31G* basis set. The time frame for the minimization of the studied compounds was due to the size of the selected individual molecule and the descriptors generated after the calculations were reported (Table 1).

Table 1. Studied phytochemicals from *Mucuna pruriens* (L.)

	Structure	**IUPAC**

1		2,3-dihydroxypropyl acetate
2		2-ethylacridine
3		4-hydroxy-2,6,6-trimethylcyclohex-2-en-1-one
4		bis(2-ethylhexyl) benzene-1,2-dicarboxylate
5		butyl 2-methylbutanoate
6		(9Z,12Z)-octadeca-9,12-dienoic acid
7		hexadecanoic acid
8		octadecanoic acid

2.2. Induce fit molecular docking analysis.

The compounds under investigation were analyzed using Molecular Operating Environment (MOE) software, where they were optimized and saved in .moe format. The relevant receptor, Phospholipase A2 (PDB ID: 1gp7), was obtained from the Protein Data Bank and prepared in MOE prior to docking analysis using the induced fit approach. Also, to verify the reliability of the engaged docking technique, the ligand obtained from the crystal structure of Phospholipase A2 (PDB ID: 1gp7) was re-docked into the active site of the same structure to assess how closely the top conformation of the re-docked ligand (i.e., the one with the lowest energy) aligned with the position of the original ligand. As a result, the similarity index between the native drug-like compound fetched from the receptor and the re-docked drug compound was comparable, and the calculated RMSD was nearly 1. Thus, the docking method applied in this study appears to be reliable.

Furthermore, the active site was identified using the site finder tool, and five distinct sites were detected, as detailed in Table 2. The first site, which exhibited the highest values across all factors, was chosen for further simulations. The computed scores were generated and documented accordingly.

Table 2. Predicted binding sites for phospholipase A_2 (PDB ID: 1gp7).

Site	Size	PLB	Hyd	Side	Amino acid residues
1	110	1.90	28	50	1:(LEU2 ILE3 PHE5 GLY6 ASN7 ILE9 GLN10 TRP19 TYR22 ALA23 TYR28 CYS29 GLY30 ALA31 GLY32 CYS45 HIS48 ASP49 ASN50 TYR52 THR53 GLN56 TYR69 PHE106)
2	10	0.07	14	20	1:(CYS44 VAL47 HIS48 CYS51 PHE94 ASN97 CYS98 VAL101 ALA102)
3	12	-0.43	10	12	1:(VAL13 PHE16 TYR22 PHE106 ALA107 ALA108, SER109 PRO110 TYR111)
4	6	-0.77	6	14	1:(LYS40 ARG43 CYS44 VAL47 CYS105 SER109)
5	4	-0.77	3	5	1:(ALA23 ASP24 CYS29 GLY30 ALA31)

2.3. Molecular dynamics simulation (MDS) study.

The molecular dynamics simulation was conducted using the GROMACS software. The compound (4-hydroxy-2,2,6-trimethylcyclohex-2-enone (compound 4)), which exhibited the highest potential to inhibit phospholipase A_2 (PDB ID: 1gp7), was further examined to evaluate its efficacy in comparison with the reference drug used in this study. The simulation was performed using the CHARMM36 m force field within the GROMACS software environment [19]. The simulation setup involved solvation of the complexes in question with appropriate water molecules and the addition of counter-ions to achieve the desired ionic concentrations. The molecular dynamics simulation was executed under the NPT ensemble, maintaining constant pressure and temperature. The simulation for the system under investigation lasted for 100 nanoseconds, and the CPPTRAJ module was used to analyze the molecular dynamics trajectories [20].

2.4. Prediction of ADMET features.

The safety and effectiveness of 4-hydroxy-2,2,6-trimethylcyclohex-2-enone (compound 4), along with the reference molecule, were evaluated for preclinical and clinical development through an ADMET study. This investigation was conducted utilizing ADMETlab software [21], taking into account various factors that were analyzed and documented.

3. Results and Discussion

3.1. Calculated 3D descriptors.

The calculated descriptors obtained from the studied compounds are reported in Table 3. The calculated highest occupied molecular orbital energy (HOMO) and lowest unoccupied molecular orbital (LUMO) energies have been observed to be regularly used to describe the chemical reaction of the studied system [22]. Also, greatest E_H value described the capability of the studied compound to release electron to the molecule with potential strength to receive from the released electron which thereby describe the compound with lowest E_L value; thus, 2-

ethylacridine (compound 2) proved to have potential greatest ability to react well and this agreed well with the work carried out by Khlood [22] (Table 4).

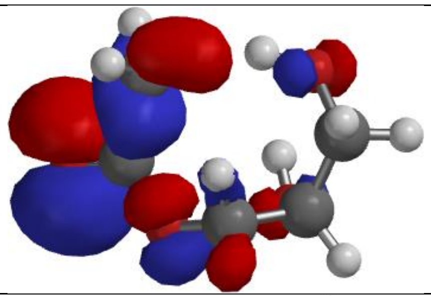
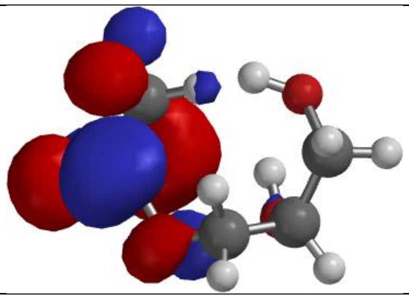
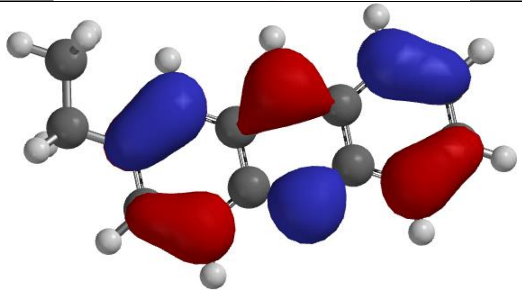
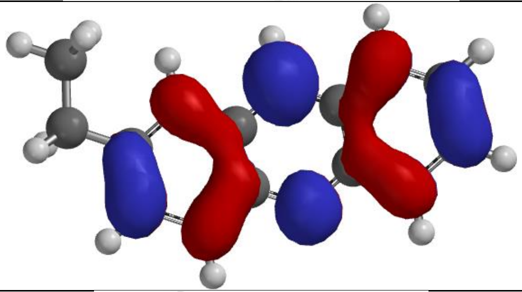
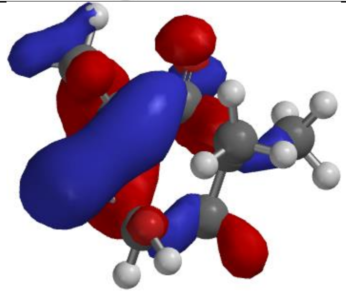
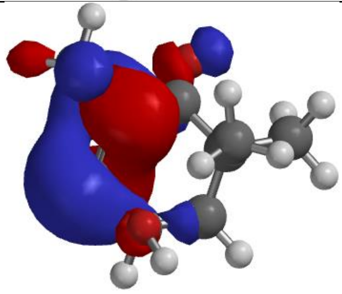
According to Ibrahim *et al*. [23], the level of steadiness of the studied compounds can be easily observed through the calculated band gap, which thereby reveals the rate of chemical reactivity in the studied system. Thus, compound 2, with an ionization potential of 3.30 eV, proved to have strong chemical reactivity. Also, other calculated descriptors were reported in Table 4.

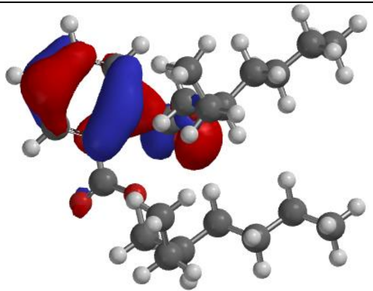
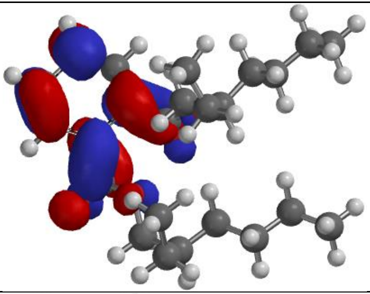
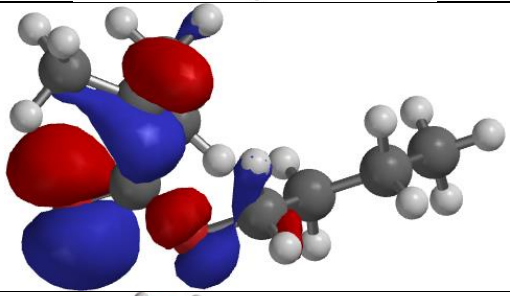
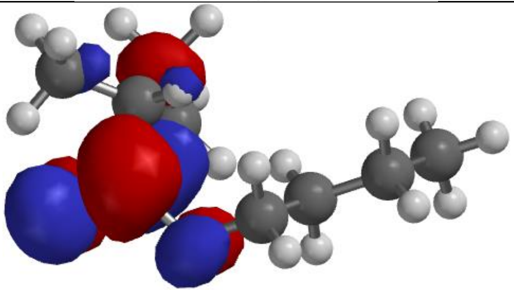
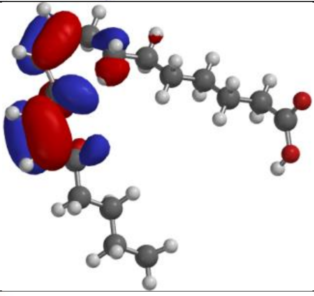
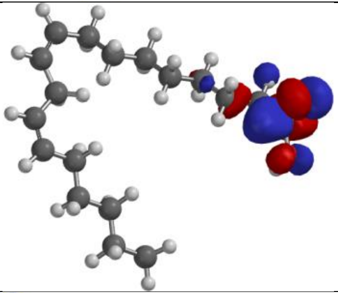
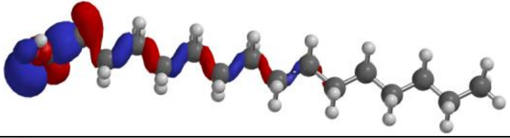
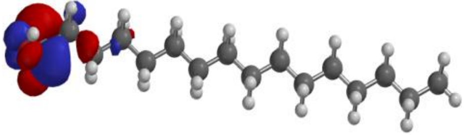
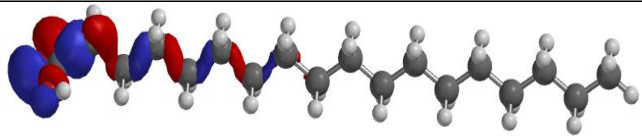
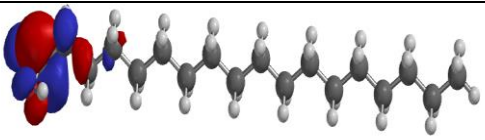
Table 3. Calculated 3-D properties of the studied/optimized compounds.

	E_H (eV)	E_L (eV)	Energy gap (eV)	Dipole moment (Debye)	Weight (Amu)	PSA (Å)	Ovality	Log P	Pol	HBD	HBA
1	-7.34	-0.38	6.96	4.96	134.131	51.933	1.21	-1.10	50.34	2	3
2	-5.38	-2.08	3.30	2.33	207.276	7.408	1.32	4.44	59.21	0	1
3	-5.50	-2.04	3.46	3.53	154.209	31.040	1.27	1.94	54.59	1	2
4	-6.91	-1.30	5.61	4.87	390.564	37.713	1.72	7.46	76.60	0	2
5	-7.02	-0.02	7.00	3.99	158.241	20.089	1.32	2.84	54.94	0	1
6	-6.09	0.05	6.14	4.26	280.452	35.189	1.62	5.97	67.83	1	1
7	-6.91	0.08	6.99	4.40	256.430	35.189	1.62	5.77	65.52	1	1
8	-7.09	0.32	7.41	4.64	284.484	34.273	1.67	6.61	68.37	1	1

PSA- polar surface area; Log P-lipophilicity; pol-polarizability; HBD-hydrogen bond donor; HBA-hydrogen bond acceptor.

Table 4. Predicted HOMO-LUMO overlay.

	HOMO	**LUMO**
1		
2		
3		

	HOMO	**LUMO**
4		
5		
6		
7		
8		

3.2. Molecular docking study.

The evaluation of the positioning of the studied molecules retrieved from *Mucuna pruriens* (L.) in the binding site of phospholipase A2 (PDB ID: 1gp7) was carefully investigated using a molecular modeling approach. Using the molecular docking method, the potential biochemical interactions between the studied ligands and the receptor were observed. Additionally, the actual binding affinity of each ligand against the receptor was calculated and presented in this work. According to reports from several literatures, biological and biochemical strength of ligand(s) may show the inhibiting ability against receptor and it may not also disclose it [24, 25]; however, Latona *et al*. reported that inhibition of receptor by ligand exposes the type of non-bonding interaction occurred between the studied complexes as well as coordination of the atom(s) in the ligands [26] and compound with lowest binding affinity is considered to be have greatest strength to inhibit receptor [27]; therefore, bis(2-ethylhexyl) benzene-1,2-dicarboxylate (Compound 4) proved to possess the ability to hinder the biochemical and biological activities of phospholipase A_2 (PDB ID: 1gp7) (Table 5). The unique inhibiting activity of compound 4 could be attributed to the release of an electron from the aromatic ring of bis(2-ethylhexyl) benzene-1,2-dicarboxylate (Compound 4) through its para-position. Additionally, the interaction point of bis(2-ethylhexyl) benzene-1,2-

dicarboxylate (Compound 4) with CYS 45 revealed that the formed complex was stable, exhibiting high efficiency and selectivity.

More so, compounds 1-3, 5, 6, and 7 proved not to be as active as the referenced compound; however, compounds 4 and 8 showed greater strength to inhibit the studied receptor than the referenced compound (Figures 1-8).

Table 5. Calculated scoring and amino acid residue.

	Binding affinity (kcal/mol)	Amino acid residues
1	-4.6303401	ASP 49 (A) H-donor
2	-5.548491	GLY 30 (A) pi-H HIS 48 (A) pi-cation
3	-4.67009783	-
4	-8.25364113	CYS 45 (A) H-donor
5	-5.51013708	HIS 48 (A) H-acceptor GLY 30 (A) H-acceptor
6	-6.98330307	ALA 31 (A) H-acceptor GLY 32 (A) H-acceptor TYR 69 (A) H-acceptor
7	-7.06063604	TYR 69 (A) H-acceptor
8	-7.62494898	ALA 31 (A) H-acceptor GLY 32 (A) H-acceptor TYR 69 (A) H-acceptor
Ref	-7.45687246	-

*Ref: Varespladib

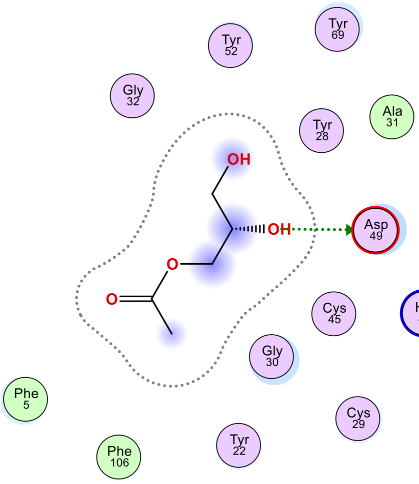


Figure 1. 2D structure of compound 1 in the active site of phospholipase A_2 (PDB ID: 1gp7).

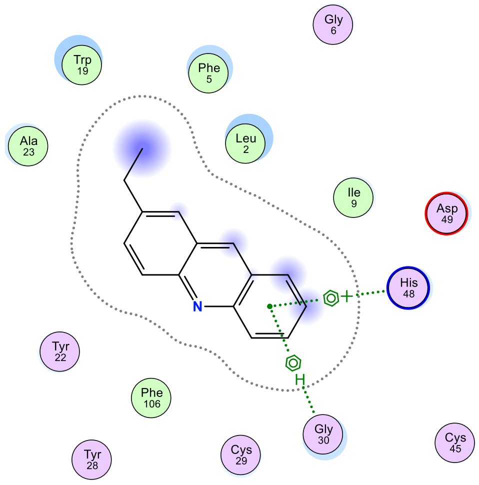


Figure 2. 2D structure of compound 2 in the active site of phospholipase A_2 (PDB ID: 1gp7).

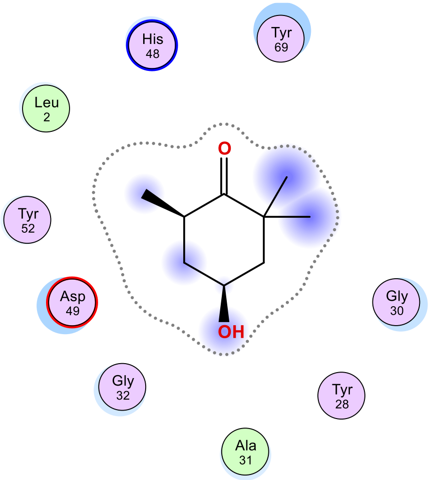


Figure 3. 2D structure of compound 3 in the active site of phospholipase A_2 (PDB ID: 1gp7).

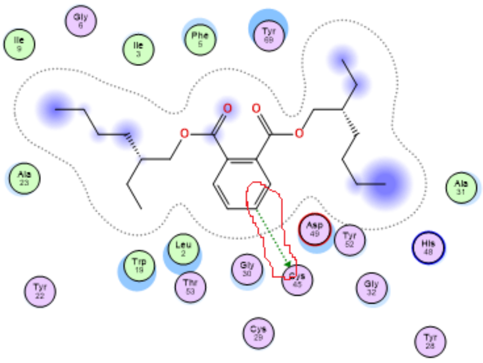


Figure 4. 2D structure of compound 4 in the active site of phospholipase A_2 (PDB ID: 1gp7).

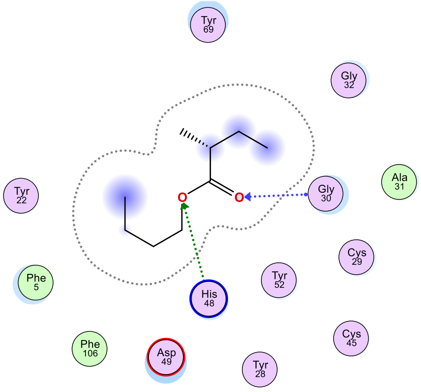


Figure 5. 2D structure of compound 5 in the active site of phospholipase A_2 (PDB ID: 1gp7).

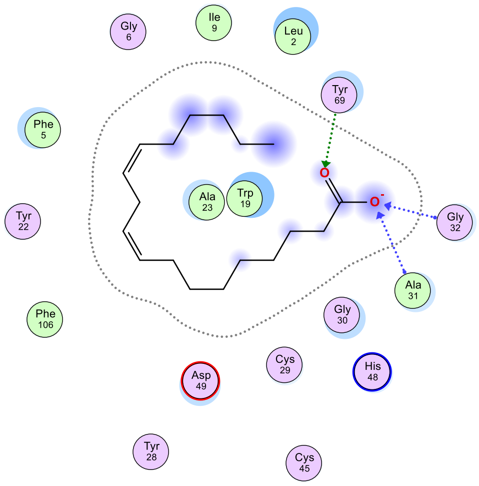


Figure 6. 2D structure of compound 6 in the active site of phospholipase A_2 (PDB ID: 1gp7).

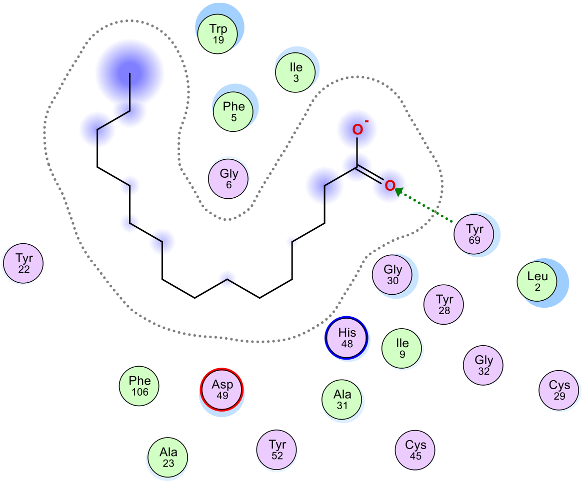


Figure 7. 2D structure of compound 7 in the active site of phospholipase A_2 (PDB ID: 1gp7).

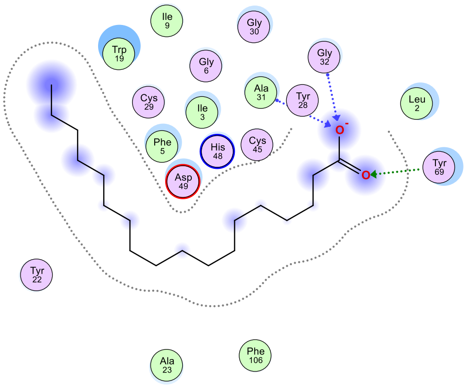


Figure 8. 2D structure of compound 8 in the active site of phospholipase A_2 (PDB ID: 1gp7).

3.3. Molecular dynamics simulation study.

3.3.1. Root mean square deviation.

The predicted root mean square deviation for compound 4 and the referenced molecule is shown in Figure 10, which formed a complex with phospholipase A_2 (PDB ID: 1gp7) during a 100,000-ps molecular dynamics simulation time. This work was conducted to demonstrate the magnitude of nonconformity of the studied ligands from the primary compound structure upon binding to the receptor and the rigidity of the studied complexes subjected to simulation. As shown in Figure 9, it was observed that Comp 4-1gp7 proved to be more stable compared to the Ref-1gp7 complex.

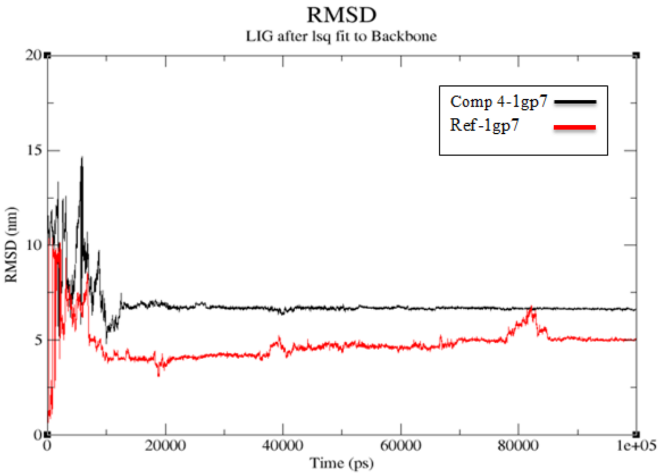


Figure 9. RMSD plot for comp4-1gp7 and ref-1gp7 complexes.

3.3.2. Root mean square fluctuation.

This section of the study revealed the flexibility of amino acid residues involved in the interaction between Comp 4-1gp7 and Ref-1gp7 during a 100,000 picosecond simulation time (Figure 10). The pattern formed by the residues involved in the Comp 4-1gp7 and Ref-1gp7 complexes in Figure 11 appears similar; nevertheless, a slight difference was observed between the configurations shown in Figure 10. Thus, a slight degree of nonconformity demonstrated by the comp 4-1gp7 complex revealed a greater degree of affinity of bis(2-ethylhexyl) benzene-1,2-dicarboxylate (compound **4**) towards phospholipase A_2 (PDB ID: 1gp7).

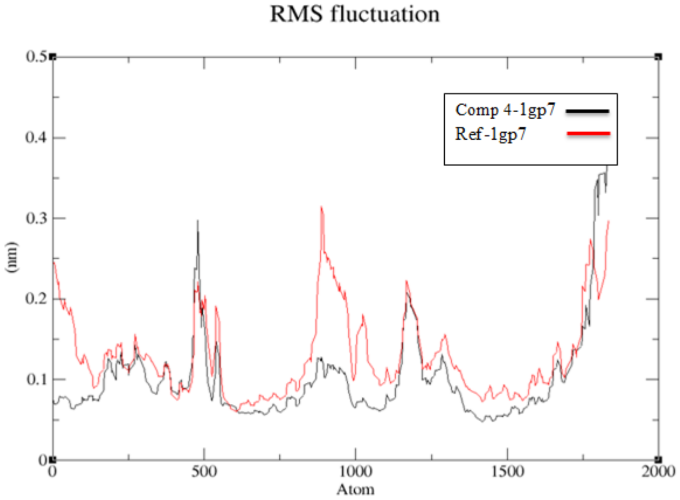


Figure 10. RMSF plot for comp4-1gp7 and ref-1gp7 complexes.

3.3.3. Calculated radius of gyration.

The calculated radius of gyration revealed the movement of the studied ligand within the active site of the receptor, which thereby inspires the structural firmness of phospholipase A2. As shown in Figure 11, the graph of the calculated radius of gyration for comp4-1gp7 initially had a low value but became stable from 60 ns to 100 ns of simulation time. It was also compared with the graph of the calculated radius of gyration for ref-1gp7. Moreover, the graph for comp4-1gp7 revealed low deviation, indicating that the comp4-1gp7 complex is in a steady state. Thus, this showed that the comp4-1gp7 complex had a great interaction during the studied simulation.

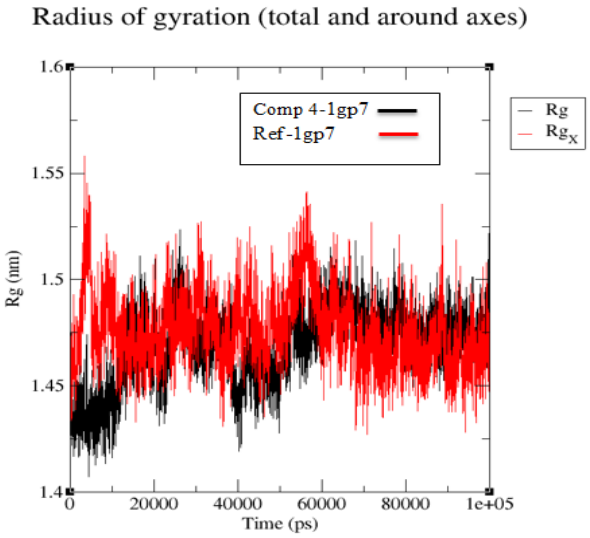


Figure 11. ROG plot for comp4-1gp7 and ref-1gp7 complexes.

3.3.4. Calculated binding energy.

The actual binding free energy was obtained for the studied complexes via MMPBSA, and the binding energy components involved were displayed in Table 6. As shown in table 6, the calculated binding energy components were ΔE_{ele} (0.07 ± 0.03 for comp4-1gp7 complex; -0.84 ± 0.09 for ref-1gp7 complex), ΔG_{gas} (0.07 ±0.03 for comp4-1gp7 complex; -0.84 ± 0.1 for ref-1gp7 complex), ΔG_{sol} (0.41 ± 0.05 for comp4-1gp7complex; 1.33 ± 0.08 for ref-1gp7 complex), and ΔG_{bind} (0.48 ± 0.02 for comp4-1gp7complex; 0.49 ± 0.02 for ref-1gp7 complex) (Figures 12 and 13). As shown in Table 6, ΔG_{sol} only favoured the inhibiting capacity of bis(2-ethylhexyl) benzene-1,2-dicarboxylate against phospholipase A_2 (PDB ID: 1gp7) compared to the inhibiting activity of the referenced compounds towards the receptor. According to the report by Oyebamiji *et al.* [30], the lower the binding free energy of a compound, the better the capacity of such a compound to interact and inhibit the target; thus, compound **4** with 0.88 kcal/mol proved to be a more potent anti-Phospholipase A_2 agent than the reference compound (Varespladib).

Table 6. Binding energy components.

Complexes	**Binding energy components (kcal/mol)**			
	ΔE_{ele}	**ΔG_{gas}**	**ΔG_{sol}**	**ΔG_{bind}**
comp4-1gp7	0.07± 0.03	0.07± 0.03	0.41± 0.05	0.48± 0.02
ref-1gp7	-0.84 ±0.09	-0.84 ±0.1	1.33 ± 0.08	0.49 ±0.02

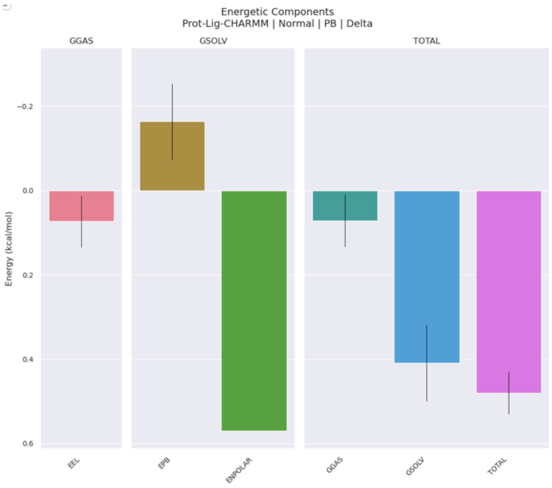


Figure 12. Chart for calculated energetic components for comp4-1gp7complex.

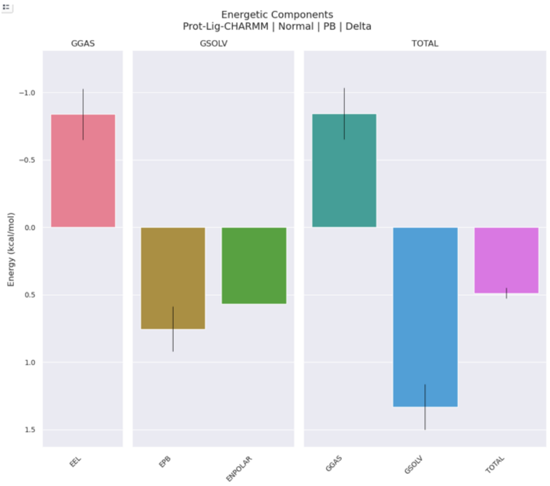


Figure 13. Chart for calculated energetic components for ref-1gp7complex.

3.4. Pharmacokinetics study.

In this work, the level of pharmacokinetic report for bis(2-ethylhexyl) benzene-1,2-dicarboxylate (Compound **4**), as well as the referenced molecule, was investigated using ADMETsar1 [29-34]. As shown in Tables 6 and 7, the calculated values for compound **4** and the referenced molecule for the brain barrier indicate that both compounds are capable of being distributed fairly throughout the brain, as evidenced by their values being greater than -1. Additionally, the human intestinal absorption values for both compounds under study were in the same range, indicating that they operate at the same rate in this regard. The predicted human intestinal absorption value for both compound 4 and *Varespladib* (Reference compound) was 0.9797 and 0.9562, respectively. The calculated values for HIA were observed to be greater than the threshold value of 30%, which indicated that the two compounds possess greater human intestinal absorption characteristics. As shown in Table 6, bis(2-ethylhexyl) benzene-1,2-dicarboxylate (Compound **4**) exhibits a potential biodegradable ability; however, the reference drug is not biodegradable (Table 7). Other calculated descriptors were obtained and reported in Tables 6 and 7.

Table 6. ADMET predicted profile --- classification for compound **4**.

Model	Result	Probability
Absorption		
Blood-Brain Barrier	BBB+	0.9383
Human Intestinal Absorption	HIA+	0.9797
Caco-2 Permeability	Caco2+	0.7003
P-glycoprotein Substrate	Substrate	0.5000
P-glycoprotein Inhibitor	Non-inhibitor	0.7174
	Non-inhibitor	0.5466
Renal Organic Cation Transporter	Non-inhibitor	0.8446
Distribution		
Subcellular localization	Mitochondria	0.8662
Metabolism		
CYP450 2C9 Substrate	Non-substrate	0.8479
CYP450 2D6 Substrate	Non-substrate	0.8655
CYP450 3A4 Substrate	Non-substrate	0.5881
CYP450 1A2 Inhibitor	Non-inhibitor	0.6539
CYP450 2C9 Inhibitor	Non-inhibitor	0.7448
CYP450 2D6 Inhibitor	Non-inhibitor	0.8432
CYP450 2C19 Inhibitor	Non-inhibitor	0.6310
CYP450 3A4 Inhibitor	Non-inhibitor	0.8309
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.6709
Excretion		
Toxicity		
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9063
	Non-inhibitor	0.7755
AMES Toxicity	Non-AMES toxic	0.9322
Carcinogens	Non-carcinogens	0.7116
Fish Toxicity	High FHMT	0.9959
Tetrahymena Pyriformis Toxicity	High TPT	0.9999
Honey Bee Toxicity	High HBT	0.5816
Biodegradation	Ready biodegradable	0.6368
Acute Oral Toxicity	IV	0.7176
Carcinogenicity (Three-class)	Warning	0.5434

ADMET Predicted Profile --- Regression

Model	Value	Unit
Absorption		
Aqueous solubility	-6.2807	LogS
Caco-2 Permeability	1.0128	LogPapp, cm/s
Distribution		
Metabolism		
Excretion		
Toxicity		

Rat Acute Toxicity	1.0838	LD_{50}, mol/kg
Fish Toxicity	0.2941	pLC50, mg/L
Tetrahymena Pyriformis Toxicity	2.1076	pIGC50, ug/L

Table 7. ADMET predicted profile --- classification for Varespladib (Reference compound).

Model	Result	Probability
Absorption		
Blood-Brain Barrier	BBB-	0.7174
Human Intestinal Absorption	HIA+	0.9562
Caco-2 Permeability	Caco2-	0.7629
P-glycoprotein Substrate	Substrate	0.7184
P-glycoprotein Inhibitor	Non-inhibitor	0.9039
	Non-inhibitor	0.5316
Renal Organic Cation Transporter	Non-inhibitor	0.8196
Distribution		
Subcellular localization	Mitochondria	0.5538
Metabolism		
CYP450 2C9 Substrate	Non-substrate	0.8138
CYP450 2D6 Substrate	Non-substrate	0.8145
CYP450 3A4 Substrate	Substrate	0.5455
CYP450 1A2 Inhibitor	Non-inhibitor	0.5872
CYP450 2C9 Inhibitor	Inhibitor	0.5414
CYP450 2D6 Inhibitor	Non-inhibitor	0.8500
CYP450 2C19 Inhibitor	Non-inhibitor	0.5059
CYP450 3A4 Inhibitor	Non-inhibitor	0.7537
CYP Inhibitory Promiscuity	High CYP Inhibitory Promiscuity	0.7159
Excretion		
Toxicity		
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9907
	Inhibitor	0.7153
AMES Toxicity	Non AMES toxic	0.7697
Carcinogens	Non-carcinogens	0.8583
Fish Toxicity	High FHMT	0.7991
Tetrahymena Pyriformis Toxicity	High TPT	0.7449
Honey Bee Toxicity	Low HBT	0.8219
Biodegradation	Not ready biodegradable	0.9723
Acute Oral Toxicity	III	0.7357
Carcinogenicity (Three-class)	Non-required	0.5138

ADMET Predicted Profile --- Regression

Model	Value	Unit
Absorption		
Aqueous solubility	-3.5742	LogS
Caco-2 Permeability	0.0547	LogPapp, cm/s
Distribution		
Metabolism		
Excretion		
Toxicity		
Rat Acute Toxicity	2.2034	LD_{50}, mol/kg
Fish Toxicity	1.3550	pLC50, mg/L
Tetrahymena Pyriformis Toxicity	0.2039	pIGC50, ug/L

ADMET- absorption, distribution, metabolism, excretion, and toxicity.

4. Conclusions

This study was conducted to analyze the actions of phytochemicals found in the selected compounds from Mucuna pruriens (L.) and to explore the non-covalent interactions between these compounds and phospholipase A2 (PDB ID: 1gp7). The descriptors obtained from the optimized phytochemicals using the 6-31G* basis set indicated the anti-venom properties of Mucuna pruriens (L.), with bis(2-ethylhexyl) benzene-1,2-dicarboxylate demonstrating the highest inhibitory effects on phospholipase A2 (PDB ID: 1gp7), as assessed through molecular modeling. Additionally, a notable correlation was found between the pharmacokinetic characteristics of bis(2-ethylhexyl) benzene-1,2-dicarboxylate and Varespladib (the reference compound).

Author Contributions

Conceptualization, A.K.O.; methodology, A.K.O. and S.A.A.; software, A.K.O., K.O.B-O; validation, S.O.A, K.A.A, and O.E.; formal analysis, A.K.O.; investigation, C.O.A. AND E.T.A; resources, A.K.O; data curation, A.K.O.; writing—original draft preparation, A.K.O., S.A.A., K.O.B-O.; writing—review and editing, A.K.O., O.E., E.T.A.; visualization, C.O.A., K.A.A.; supervision, E.T.A. and C.O.A.; project administration, A.K.O. and O.E.; funding acquisition, C.O.A. All authors have read and agreed to the published version of the manuscript.

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Not applicable.

Data Availability Statement

Data supporting the findings of this study are available upon reasonable request from the corresponding author.

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Nil

Conflict of Interest

Authors declare no conflict of interest.

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