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In-Silico Evaluation of Moringa Oleifera Phytochemicals as Inhibitors Against Newcastle Disease Virus in Birds

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ABSTRACT

Newcastle disease (ND) is a highly contagious viral infection that significantly impacts the global poultry industry, leading to substantial economic losses due to high mortality rates. Despite the availability of vaccines, the persistent threat of ND necessitates the exploration of novel therapeutic strategies. Moringa oleifera, commonly known as the "miracle tree," is renowned for its diverse pharmacological properties, including antiviral, antioxidant, and immunomodulatory effects. This study investigates the potential of Moringa oleifera phytochemicals as inhibitors of the Newcastle disease virus (NDV) through molecular docking analysis.

The research focuses on identifying bioactive compounds within Moringa oleifera that exhibit strong binding affinities to the fragment of fusion protein of NDV, a critical component in the virus's ability to infect host cells. By employing in silico methods, the study predicts the efficacy of these compounds in disrupting NDV activity, offering a promising new avenue for ND treatment and prevention. The findings suggest that specific phytochemicals, such as Beta Sitosterol, catechins, and Kaempferol, demonstrate significant binding affinities, (with binding scores of -8.5 kcal/mol, -8.2 kcal/mol and -7.9 kcal/mol respectively), making them potential candidates for further development into antiviral therapies.

This approach underscores the potential of Moringa oleifera in enhancing the resilience of the poultry industry against Newcastle disease, thereby contributing to improved food security and economic stability.

Keywords: Newcastle disease, Moringa oleifera, Molecular docking, Fusion protein, Poultry industry,

INTRODUCTION

Newcastle disease (ND) is a highly contagious and severe viral infection caused by the Newcastle disease virus (NDV). Since its discovery in Newcastle, United Kingdom in 1926, this disease has had a negative impact on poultry industries and other wild bird species in a loss of productivity and affects economy [1]. The disease has quickly spread over the world, becoming widespread in many nations where agriculture is the primary source of wealth [2]. A recent report suggest it can be found in vaccinated poultry houses [3]. Newcastle disease virus (NDV), also known as avian paramyxovirus serotype 1 (APMV-1), is responsible for the infection [4]. NDV belongs to the genus Avulavirus from the Paramyxoviridae family. The virus genome is a single-stranded, negative sense, non-segmented RNA molecule of approximately

15.2 kb that encodes six structural proteins: nucleoprotein (NP), phosphoprotein (P), matrix protein (M), fusion (F), hemagglutinin-neuraminidase (HN), and RNA polymerase (L) [5], [6], [7]. Previously categorized in the Rubulavirus genus, NDV is now recognized as an Avulavirus in the Paramyxovirinae subfamily. Family Paramyxoviridae. APMV-1, APMV-2, and APMV-3 are three of the ten avian paramyxovirus serotypes that cause major illness in chicken [8]. The virion's envelope is formed from the host cell's plasma membrane and is made up of two viral glycoproteins (8-12 nm): fusion (F) and hemagglutinin-neuraminidase (HN). The F protein connects the viral envelope to the host cell membrane, whereas the HN protein links the virus to the receptor. The F and HN proteins are the primary immunogenic proteins, [1]. The virus enters the host cell through surface glycoproteins (HN and F). The HN protein attaches to sialic acid-containing cell receptors on the host cell's surface, whereas the F protein, after the viral envelope fuses with the host cell membrane, the viral nucleocapsid is released into the cytoplasm, where the transcription and translation phases begin. The negative-sense RNA genome is transcribed into positive-sense mRNA, which is then translated into viral protein [9], [10]. Because of the high mortality rate (100%) caused by new castle diseases (ND) in chicken sectors, trade restrictions and embargoes have had a substantial impact on the places where it has spread. The use of vaccines can help to reduce the catastrophic effects of NDV. Many commercial vaccinations, both live and dead, are available on the market [11], [12].

Moringa oleifera is also known as the "miracle tree" due to its numerous applications and adaptability. [13]. It is widely cultivated around the world due to its nutritional and medicinal properties [14]. various part of Moringa oleifera such as leaves, pods, stem flower and roots are used by some countries like Africa, Hawaii, Philippines, India and Pakistan as vegetable Africa [15], ([16]. All part of Moringa oleifera contained crotenoids, alkaloids, flavonoids, glycosides, anthocyanin, anthraquinonoids, saponins, steroids, tannins, and terpenoids [17]. These phytochemicals presence in Moringa oleifera contribute in the treatment of various infections like cancer, diabetes, cardiovascular diseases, age related disorder, [18]. it also reported that phytochemicals presence in Moringa oleifera can be used for the curing of diseases like arthritis, inflammations, Antioxidants, Anticancer, Antimicrobial, antiviral, and antidiabetic-properties [15], [17].

Molecular docking is a commonly used computational approach in the field of molecular recognition research to forecast both binding affinity and scores. [18]. The simulation of the docking process is a complicated process in this approach, the protein and the ligand are separated by some physical distance, and the ligand finds its position into the protein’s active site after a certain number of “moves” in its conformational space [29]. The advancement in bioinformatics, systems biology, and techniques in computer brought about changes in the field of pharmacy allowing for the discoveries, development and designed of effectives pharmaceuticals products for the effective therapeutic application, Studies have shown that’s, it’s possible to forecast and determine the binding affinity of a leads molecule that can precisely bind a receptor for the treatment of certain diseases using computational program and software [20]. The PyRx virtual screening tool AutoDock Vina is used to conduct molecular docking studies [21].

In the present study, molecular docking analysis will employed to determine the binding conformation of the crystal structure of the receptor protein using the phytochemicals of Moringa oleifera plant. This work will utilize the use of NDV strain LaSota (LS-wt) which is used worldwide as a live or inactivated vaccine and was used here as a control vaccine in the immunization challenge experiments for standard inhibitors of the aforesaid disease. The purpose of the study or research is Identification of Active Compounds which

Determine the specific phytochemicals present in Moringa oleifera that have potential inhibitory effects on the fragment fusion protein of new castle diseases

MATERIALS AND METHODS

Library preparation of phytochemical

Swiss ADME and FAF Drugs 4 software Phytochemical Screening

A 30 Phytocompound from Moringa oleifera plant that have bioactivity related to virus was selected and screened using online Swiss ADME and FAF Drugs 4 software's via (http://www.swissadme.ch), visions and FAFDrugs4(univ-paris-diderot.fr) Vision 2.7 to predict the pharmacokinetics and drugs likeness bioavailability, Absorption rate, distribution, metabolisms, excretion, toxicity and structure activity of phytochemicals in the animals body.

Pass online and adverse and toxic effects software screening of phytochemicals

Moringa oleifera phytocompound was also screened using Pass online and adverse effects software via (https://www.way2drug.com › pass online) to predicts the biological activity, activity spectrum, drugs discovery, virtual screening and potential therapeutic and some adverse effects of the micro molecule of the phytocompound

SEA Search Server software screening

The phytocompound of Moringa oleifera was also screened using SEA Search server software via (https://sea.bkslab.org), while predicting or exploration of similarities between protein targets and small molecule ligands of new castle disease such as protein targets identification, ligand similarities, virtual screening and structural activity relationship of the protein (fusion protein [1G5G]) and provide comprehensive analyses of protein-ligand interactions, including docking studies and binding affinity predictions

DIGEP Pred software screening

The phytocompound of Moringa oleifera have being screened using DIGEP Pred online software via (https://www.way2drug.com) vision 2.0 for the predictions of Its predictive capabilities provide valuable insights into upregulation and downregulation of certain protein biological activities and aid in experimental design for further validation and analysis.

Protein preparation

The PDB structures of important viral proteins (Fragment of fusion protein) related to Avian new castles diseases was download from the Protein Data Bank in SDF Format, via (https://www.rcsb.org) avian paramyxovirus serotype 1 (APMV-1), using (PDB entry code: 1G5G which was converted to pdbqt format using PYMOL Software https://pymol.orgsales@schrodinger) vision 3.0.3 and all natural ligand and water was removed, hydrogen, charges ware added using Autodock tool vision 1.5.7 for the docking simulation activities

Phytochemical library preparation

The literature-based 3D or 2D structure of natural product compounds was accessed and Downloaded from the phytocompound databases PubChem using (PubChem (nih.gov), created a library of phytochemicals of Moringa oleifera in SDF format and converted to pdbqt format using PYMOL Software (https://pymol.orgsales@schrodinger) vision 3.0.3 which is used for docking simulation.

Molecular docking

Molecular docking simulations was conducted using molecular docking software (auto doc vina), (http://vina.scripps.edu) vision 1.5.7 phytochemicals interactions (hydrogen bonds, hydrophobic

interactions) and binding affinities (binding energy scores) was conducted with chosen viral targets, Docking analysis was visualized and evaluated, possible lead compounds and Visualization of docking interaction was conducted using discovery studio (viewer) (https://en.wikipedia.org › wiki › Discovery Studio) vision 20.1.0.0.

RESULTS

Table 1 Physiochemical and Pharmacokinetic Properties of Chemical Compounds Using (SWISS ADME AND FAFDrugs4)

Compound Name	Moringine	Morumoside A	Beta Sitosterol	Quercetin	Kaempferol	catechins,
MW	107.15	297.3	414.71	302.24	286.24	290.27
Heavy atoms	8	21	30	22	21	21
Fraction Csp3	0.14	0.5	0.93	0	0	0.2
Rotatable bonds	1	4	6	1	1	1
H-bond acceptors	1	6	1	7	6	6
H-bond donors	1	4	1	5	4	5
MR	34.12	72.11	133.23	78.03	76.01	74.33
XLOGP3	1.09	-1.21	9.34	1.54	1.9	0.36
WLOGP	0.99	-1.08	8.02	1.99	2.28	1.22
ESOL Solubility (mg/ml)	2.24E+00	4.03E+01	5.23E-06	2.11E-01	1.40E-01	1.74E+00
GI absorption	High	High	Low	High	High	High
BBB permeant	Yes	No	No	No	No	No
Lipinski violations	0	0	1	0	0	0
Ghose violations	3	1	3	0	0	0
Veber violations	0	0	0	0	0	0

Egan violations	0	0	1	0	0	0
Muegge violations	2	0	2	0	0	0
Bioavailability Score	0.55	0.55	0.55	0.55	0.55	0.55
Lead likeness violations	1	0	2	0	0	0
Synthetic Accessibility	1	4.03	6.3	3.23	3.14	3.5
Solubility(mg/l)	26790.11	137933.41	153.84	15228.15	12543.68	33856.42
Solubility Forecast Index	Good	Good	Good	Good	Good	Good
Oral_Bioavailability_VEBER	Solubility	Solubility	Solubility	Solubility	Solubility	Solubility
Oral_Bioavailability_EGAN	Good	Good	Good	Good	Good	Good
Traffic Lights	Good	Good	Good	Good	Good	Good
Acceptability	Accepted	Accepted	Accepted	Accepted	Accepted	Accepted
Total Charge	1	0	0	0	0	0

Table 1 above is an integrated table of SwissADME and FAF-Drugs4 tables that provides a thorough overview of a compound's physicochemical and pharmacokinetic properties, which is required for determining its potential as a drug candidate. The physical parameters include molecular weight, lipophilicity (XLogP3), and water solubility, all of which affect a compound's bioavailability, membrane permeability, and overall drug-likeness. The table also includes hydrogen bond donors and acceptors, as well as rotatable bonds that influence the compound's flexibility and interaction with biological targets. On the pharmacokinetic side, the merged table provides all available data about ADMET (absorption, distribution, metabolism, excretion, and Toxicity) features. It evaluates parameters like gastrointestinal absorption, blood-brain barrier penetration, and interactions with fragment of fusion protein (1G5G), which are critical for predicting a compound's behaviour in the body. SwissADME and FAF-Drugs4 analysis give a comprehensive profile for evaluating a compound's potential as a drug candidate, balancing efficacy, safety, and bioavailability

Table 2 Biological Activities of Chemical Compounds (pass online)

		Biological activities (Pa>Pi .Pa>0.7)		
S/N	**Compound name**	**Pa**	**Pi**	**Activity**
1	Moringine	0.911	0.004	Phobic disorders treatment
		0.871	0.003	Venombin AB inhibitor
		0.865	0.003	Threonine aldolase inhibitor
		0.859	0.004	Complement factor D inhibitor
		0.858	0.003	Polyamine-transporting ATPase inhibitor
		0.865	0.016	Aspulvinone dimethylallyltransferase inhibitor
		0.854	0.007	Glucose oxidase inhibitor
		0.848	0.004	Fusarinine-C ornithinesterase inhibitor
		0.847	0.005	Arginine 2-monooxygenase inhibitor
		0.848	0.012	Methylenetetrahydrofolate reductase (NADPH) inhibitor
		0.839	0.005	Omptin inhibitor
		0.836	0.004	NADPH-cytochrome-c2 reductase inhibitor
		0.794	0.004	Aspartate-ammonia ligase inhibitor
		0.795	0.007	2-Hydroxymuconate-semialdehyde hydrolase inhibitor
		0.734	0.004	Glucan 1,4-alpha-maltotetraohydrolase inhibitor
2	Morumoside A	0.945	0.004	CDP-glycerol glycerophosphotransferase inhibitor
		0.930	0.005	Membrane integrity agonist
		0.905	0.003	Vasoprotector
		0.857	0.002	Lactase inhibitor
		0.831	0.012	Sugar-phosphatase inhibitor
		0.753	0.004	Fructan beta-fructosidase inhibitor
		0.758	0.009	UDP-N-acetylglucosamine 4-epimerase inhibitor
		0.744	0.011	Oxidoreductase inhibitor
		0.757	0.005	Antiinfective
		0.734	0.002	Beta-D-fucosidase inhibitor
		0.740	0.011	Antidyskinetic
		0.704	0.003	Mannose isomerase inhibitor
		0.703	0.002	4-Alpha-glucanotransferase inhibitor
		0.705	0.008	Levanase inhibitor
3	Beta Sitosterol	0.965	0.001	DELTA14-sterol reductase inhibitor
		0.960	0.002	Antihypercholesterolemic

		0.959	0.002	Prostaglandin-E2 9-reductase inhibitor
		0.957	0.001	Cholesterol antagonist
		0.945	0.002	Alkylacetylglycerophosphatase inhibitor
		0.888	0.002	UGT2B substrate
		0.889	0.003	UDP-glucuronosyltransferase substrate
		0.886	0.003	Oxidoreductase inhibitor
		0.881	0.004	Anesthetic general
		0.855	0.004	Linoleate diol synthase inhibitor
		0.849	0.006	Respiratory analeptic
		0.847	0.003	Cholestanetriol 26-monooxygenase inhibitor
		0.796	0.010	Protein-disulfide reductase (glutathione) inhibitor
		0.788	0.002	N-(long-chain-acyl)ethanolamine deacylase inhibitor
		0.788	0.005	Glucan endo-1,3-beta-D-glucosidase inhibitor
4	Quercetin	0.973	0.002	Membrane integrity agonist
		0.969	0.002	HIF1A expression inhibitor
		0.962	0.001	Peroxidase inhibitor
		0.957	0.002	HMOX1 expression enhancer
		0.938	0.003	Membrane permeability inhibitor
		0.895	0.002	Histidine kinase inhibitor
		0.894	0.002	UGT1A3 substrate
		0.891	0.001	Iodide peroxidase inhibitor
		0.894	0.004	Aldehyde oxidase inhibitor
		0.887	0.002	AR expression inhibitor
		0.797	0.012	Antineoplastic
		0.789	0.004	Sulfotransferase substrate
		0.788	0.004	Lipid peroxidase inhibitor
		0.770	0.001	3-Oxoacyl-[acyl-carrier-protein] synthase inhibitor
5	Kaempferol	0.983	0.001	Chlordecone reductase inhibitor
		0.974	0.002	Membrane integrity agonist
		0.969	0.002	HIF1A expression inhibitor
		0.965	0.001	2-Dehydropantoate 2-reductase inhibitor
		0.961	0.001	Aryl-alcohol dehydrogenase (NADP+) inhibitor
		0.896	0.003	CYP1A inhibitor
		0.894	0.002	Antihemorrhagic
		0.894	0.004	Anaphylatoxin receptor antagonist
		0.890	0.002	Xenobiotic-transporting ATPase inhibitor
		0.858	0.001	Testosterone 17beta-dehydrogenase inhibitor
		0.798	0.001	Alcohol dehydrogenase [NAD(P)+] inhibitor
		0.797	0.002	NOS2 expression inhibitor

		0.794	0.005	27-Hydroxycholesterol 7alpha-monooxygenase inhibitor
		0.782	0.003	UGT1A8 substrate
		0.783	0.004	Lipid peroxidase inhibitor
6	catechins,	0.983	0.001	Membrane integrity agonist
		0.962	0.003	Mucomembranous protector
		0.959	0.003	TP53 expression enhancer
		0.939	0.002	HMOX1 expression enhancer
		0.927	0.002	Sulfotransferase substrate
		0.888	0.003	Lipid peroxidase inhibitor
		0.875	0.004	UDP-glucuronosyltransferase substrate
		0.877	0.007	Chlordecone reductase inhibitor
		0.863	0.003	APOA1 expression enhancer
		0.848	0.003	Histidine kinase inhibitor
		0.795	0.005	Anticarcinogenic
		0.791	0.003	Histamine release inhibitor
		0.795	0.010	Aldehyde oxidase inhibitor
		0.789	0.005	UGT1A substrate
		0.788	0.004	Chemopreventive
		0.787	0.004	P-benzoquinone reductase (NADPH) inhibitor
		0.790	0.011	Membrane permeability inhibitor
		0.785	0.009	JAK2 expression inhibitor
		0.755	0.001	Glutathione-disulfide reductase inhibitor
		0.743	0.009	Kinase inhibitor

Table 2 presents a focused analysis of the predicted biological activities of various compounds using PASS (Prediction of Activity Spectra for Substances) software, with emphasis placed on activities where the probability of activity (Pa) is greater than the probability of inactivity (Pi) and Pa is greater than 0.7. Pa > 0.7 implies a high probability that the chemical will exhibit the expected biological activity. This table presents data on phytocompounds that are expected to have a major influence on specific biological targets. The data helps improve the drug discovery process by identifying chemical compounds that are more likely to be useful in biological systems. The table can help identify the most promising candidates for subsequent biological testing and drug development

Table 3 Possible Adverse & Toxic Effects of Chemical Compounds.

		Possible Adverse & Toxic Effects (Pa>Pi. Pa>0.7)		
S/No.	**Compound Name**	**Pa**	**Pi**	**Activity**
1	Moringine	0.914	0.005	Acidosis, metabolic
		0.869	0.011	Pure red cell aplasia
		0.862	0.013	Twitching
		0.847	0.005	Multiple organ failure

		0.843	0.005	Nail discoloration
		0.836	0.015	Neutrophilic dermatosis (Sweet's syndrome)
		0.820	0.019	Euphoria
		0.802	0.005	Hypomagnesemia
		0.798	0.009	Galactorrhea
		0.790	0.004	Anemia, sideroblastic
		0.783	0.004	Acneiform eruption
		0.772	0.004	Adrenal cortex hypoplasia
		0.775	0.010	Weight gain
		0.719	0.011	Respiratory impairment
		0.731	0.024	Postural (orthostatic) hypotension
2	Morumoside A	0.851	0.003	Hypercholesterolemic
		0.805	0.014	Inflammation
		0.800	0.021	Drowsiness
		0.784	0.005	Ototoxicity
		0.792	0.019	Thrombocytopenia
		0.795	0.026	Diarrhoea
		0.791	0.025	Behavioral disturbance
		0.789	0.028	Hematotoxic
		0.786	0.028	Toxic, gastrointestinal
		0.782	0.026	Nausea
		0.774	0.021	Weakness
		0.777	0.026	Emetic
		0.764	0.020	Excitability
3	Beta Sitosterol	0.928	0.008	Sleep disturbance
		0.904	0.010	Conjunctivitis
		0.896	0.005	Teratogen
		0.890	0.005	Embryotoxic
		0.876	0.010	Reproductive dysfunction
		0.869	0.011	Ocular toxicity
		0.841	0.003	Hypercholesterolemic
		0.848	0.010	Inflammation
		0.777	0.005	Cholestasis
		0.782	0.017	Hypertensive
		0.781	0.023	Headache
		0.784	0.028	Toxic, gastrointestinal
		0.779	0.024	Pain
		0.764	0.020	Excitability

		0.744	0.021	Necrosis
4	Quercetin	0.797	0.018	Toxic, vascular
		0.766	0.052	Shivering
		0.706	0.014	Endocrine disruptor
		0.719	0.032	Reproductive dysfunction
5	Kaempferol	0.843	0.003	Genotoxic
		0.773	0.023	Toxic, vascular
		0.783	0.044	Shivering
		0.702	0.024	Inflammation
		0.710	0.040	Hematotoxic
6	catechins	0.810	0.014	Inflammation
		0.789	0.020	Toxic, vascular
		0.786	0.017	Hypertensive
		0.791	0.026	Diarrhoea
		0.769	0.011	Panic
		0.751	0.023	Neurotoxic
		0.740	0.022	Nephrotoxic
		0.768	0.051	Shivering
		0.715	0.016	Withdrawal

Table 3 will discuss on the possible adverse and toxic effects that a chemical compound may have when the probability of its activity (Pa) is greater than 0.7. The table's data will be an invaluable resource for identifying compounds that are very likely to have adverse effects, which will be extremely important in drug development. Remember that Pa ≠ Pi and Pa > 0.7.

Table 4 Target Molecules for Chemical Compounds Similarity Ensemble Approach using sea search server software.

S/No.	Query Molecule	Target Key	Target Name	P-Value	MaxTC	Description
1	Moringine	LOXL2_HUMAN	LOXL2	3.73E-71	0.5	Lysyl oxidase homolog 2
		CBPA3_HUMAN	CPA3	7.61E-54	0.31	Mast cell carboxypeptidase A
		TAAR1_MACMU	TAAR1	1.63E-47	0.5	Trace amine-associated receptor 1

		S15A1_HUMAN	SLC15A1	8.65E-45	0.32	Solute carrier family 15 member 1
		CXCR4_RAT	Cxcr4	1.45E-25	0.43	C-X-C chemokine receptor type 4
		AOC3_BOVIN	AOC3	4.60E-20	0.39	Membrane primary amine oxidase
		GABT_PIG	ABAT	1.51E-19	0.32	4-aminobutyrate aminotransferase, mitochondrial
		GGPPS_YEAST	BTS1	2.42E-18	0.29	Geranylgeranyl pyrophosphate synthase
		HISX_ECOLI	hisD	2.95E-17	0.28	Histidinol dehydrogenase
		C11B1_BOVIN	CYP11B1	1.74E-06	0.28	Cytochrome P450 11B1, mitochondrial
2	Morumoside A	SC5A2_MOUSE	Slc5a2	3.99E-65	0.33	Sodium/glucose cotransporter 2
		B4GT1_BOVIN	B4GALT1	5.04E-47	0.32	Beta-1,4-galactosyltransferase 1
		FIMH_ECOLI	fimH	6.24E-44	0.42	Type 1 fimbrin D-mannose specific adhesin
		S28A3_HUMAN	SLC28A3	1.13E-35	0.29	Solute carrier family 28 member 3
		A0MJA4_CANLF	NPC1L1	2.06E-24	0.31	Niemann-Pick C1-like 1 protein
		O96394_LEIAM		1.35E-19	0.36	Arginase

		ECE1_BOVIN	ECE1	1.88E-08	0.31	Endothelin-converting enzyme 1
		PPO2_AGABI	PPO2	1.19E-07	0.37	Polyphenol oxidase 2
		Q1R2J4_ECOUT	fimH	1.55E-12	0.29	Type 1 fimbiral adhesin FimH
		ECE1_BOVIN	ECE1	1.88E-08	0.31	Endothelin-converting enzyme 1
3	Beta Sitosterol	VDR_PIG	VDR	9.85E-37	0.3	Vitamin D3 receptor
		CP125_MYCTU	cyp125	1.00E-86	0.72	Steroid C26-monooxygenase
		CP17A_HUMAN	CYP17A1	6.61E-86	0.68	Steroid 17-alpha-hydroxylase/17,20 lyase
		Q9GRG7_9TRYP	g6pd	1.93E-59	0.48	Glucose-6-phosphate 1-dehydrogenase
		CP17A_MACFA	CYP17A1	3.86E-55	0.46	Steroid 17-alpha-hydroxylase/17,20 lyase
		VDR_RAT	Vdr	1.44E-50	0.33	Vitamin D3 receptor
		VDR_CHICK	VDR	1.50E-22	0.3	Vitamin D3 receptor
		ERG2_YEAST	ERG2	8.51E-20	0.43	C-8 sterol isomerase
		CAH4_BOVIN	CA4	7.70E-30	0.38	Carbonic anhydrase 4

		REST_BORBU	resT	7.45E-14	0.35	Telomere resolvase ResT
		ABCBB_HUMAN	ABCB11	8.44E-14	0.35	Bile salt export pump
4	Quercetin	LOX15_RABIT	ALOX15	4.80E-11	1	Arachidonate 15-lipoxygenase
		AMPC_ECOLI	ampC	3.95E-06	1	Beta-lactamase
		LOX15_HUMAN	ALOX15	1.35E-10	1	Arachidonate 15-lipoxygenase
		FAS_CHICK	FASN	8.47E-66	0.55	Fatty acid synthase
		TRY1_BOVIN		0.02911	1	Cationic trypsin
		CP2C9_HUMAN	CYP2C9	0.3759	1	Cytochrome P450 2C9
		EGFR_HUMAN	EGFR	0.5613	1	Epidermal growth factor receptor
		CAH9_HUMAN	CA9	0.1696	1	Carbonic anhydrase 9
		FAK1_HUMAN	PTK2	0.2365	1	Focal adhesion kinase 1
		VE6_HPV16	E6	1.05E-108	0.76	Protein E6
5	Kaempferol	FAS_CHICK	FASN	3.50E-52	0.43	Fatty acid synthase
		OPRD_PIG	OPRD1	1.42E-51	0.43	Delta-type opioid receptor
		BGLR_RAT	Gusb	2.35E-61	0.57	Beta-glucuronidase

		O96394_LEIAM		3.75E-77	0.78	Arginase
		PTPRS_HUMAN	PTPRS	2.01E-59	0.75	Receptor-type tyrosine-protein phosphatase S
		DDL_HELPH	ddl	3.69E-94	0.78	D-alanine--D-alanine ligase
		AK1CL_MOUSE	Akr1c21	3.69E-94	0.78	Aldo-keto reductase family 1 member C21
		FPS_FUJSV	V-FPS	3.87E-90	0.75	Tyrosine-protein kinase transforming protein Fps
		VE6_HPV16	E6	1.67E-114	0.75	Protein E6
		CP2D6_HUMAN	CYP2D6	0.7323	1	Cytochrome P450 2D6
6	catechins,	PGH1_SHEEP	PTGS1	3.22E-09	1	Prostaglandin G/H synthase 1
		NOS3_BOVIN	NOS3	1.30E-08	1	Nitric oxide synthase, endothelial
		CAH3_HUMAN	CA3	4.83E-13	1	Carbonic anhydrase 3
		Q8I2J3_PLAF7		1.10E-05	1	M18 aspartyl aminopeptidase
		NANH_CLOPF	nanH	9.32E-07	0.31	Sialidase
		LOX15_RABIT	ALOX15	1.72E-07	0.44	Arachidonate 15-lipoxygenase
		DHPR_RAT	Qdpr	3.39E-10	0.31	Dihydropteridine reductase
		FABI_ECOLI	fabI	1.75E-12	0.6	Enoyl-[acyl-carrier-protein] reductase [NADH] FabI

		MTSI_SPISQ	sssIM	1.11E-16	0.47	CPG DNA methylase

The data in Table 4 was obtained using the Sea search server and Similarity Ensemble Approach softwares. The resulting table includes a list of probable target proteins and macromolecules with which the given compounds may interact, as well as the probability of each phytocompounds action on its target. The table's data will help to hasten the identification of promising drug candidates, as well as the development of targeted therapies.

Table 5 Cellular Gene Expressions Induce by Chemical Compounds.

S/N	**Compound Name**	**Pa**	**Pi**	**Genes**	**Gene Expression**		
1	Moringine	**Pa**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.816	0.024	TEP1	0.781	0.025	HTATIP2
		0.75	0.032	ACSL3	0.79	0.056	SAT
		0.727	0.027	DNASE2	0.756	0.033	IFIT1
		0.747	0.058	SREBF2	0.736	0.02	TPI1
		0.719	0.038	AMHR2	0.786	0.079	POR
		0.725	0.056	IFI27	0.744	0.074	MGST1
		0.701	0.042	FABP4	0.727	0.072	C1ORF63
					0.703	0.069	VNN1
					0.709	0.115	AKR1B10
2	Morumoside A	**Pa**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.906	0.006	ADAM19	0.875	0.01	TCF12
		0.882	0.014	MAPK8	0.849	0.007	KDELC1
		0.85	0.009	SDC4	0.849	0.007	PGM2
		0.841	0.012	APTX	0.778	0.03	SMPD1
		0.841	0.012	RSBN1			
		0.813	0.012	SYMPK			
		0.807	0.02	EVI2B			
		0.811	0.026	OSBP2			
		0.793	0.038	PDZK1			
		0.766	0.022	FKBP5			
		0.797	0.06	DMXL1			
		0.738	0.011	CXCL10			
3	Beta Sitosterol	**Pa**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.908	0.019	MAST4	0.92	0.022	CACNG4
		0.908	0.019	MSH5	0.92	0.022	CHRNE
		0.908	0.019	TREX1	0.897	0.005	WISP2
		0.905	0.022	CBX2	0.908	0.019	KCNH2
		0.904	0.023	RPS19	0.908	0.019	PLLP
		0.912	0.031	TMSB15A	0.908	0.019	SLC25A4

		0.9	0.023	ADORA2B	0.908	0.019	ZYX
		0.9	0.023	CDCA4	0.906	0.022	PPP1CB
		0.9	0.023	CENPJ	0.905	0.022	TMEM56
		0.9	0.023	DNAJC9	0.9	0.023	PFKFB2
		0.9	0.023	DONSON	0.9	0.023	SH3BGRL
		0.9	0.023	HAUS8	0.9	0.023	TRIM13
		0.9	0.023	MCMBP	0.892	0.023	CREB3L1
		0.9	0.023	NUP155	0.892	0.023	RNASEL
		0.9	0.023	PARP2	0.896	0.033	IL4R
		0.9	0.023	RMI1	0.887	0.028	COL22A1
		0.9	0.023	SCARA3	0.887	0.028	KLK13
		0.9	0.023	SLC16A14	0.882	0.029	CNN2
		0.9	0.023	SUV39H2	0.882	0.029	DYSF
		0.89	0.026	C9ORF40	0.882	0.029	FAM110C
		0.882	0.029	CTSL2	0.882	0.029	MYO5B
4	Quercetin	**A**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.968	0.003	IFIH1	0.966	0.002	GNAI1
		0.966	0.002	COG5	0.966	0.002	LIPH
		0.966	0.002	DNAAF2	0.966	0.002	PITPNM1
		0.966	0.002	FARP1	0.96	0.003	KHNYN
		0.966	0.002	FCHSD2	0.959	0.002	GCKR
		0.966	0.002	KIAA0182	0.959	0.004	CITED2
		0.966	0.002	LGALS8	0.948	0.003	CEP72
		0.966	0.002	MAL2	0.947	0.003	VNN1
		0.966	0.002	METTL10	0.948	0.003	RGL1
		0.966	0.002	MOGAT2	0.948	0.003	SLC39A4
		0.966	0.002	SATB2	0.949	0.006	FAM171A1
		0.967	0.004	DMXL1	0.949	0.006	HSPA12A
		0.964	0.002	PLSCR4	0.949	0.006	LIM2
		0.963	0.003	SERPINB1	0.949	0.006	MAP3K7IP3
		0.961	0.003	H1FX	0.949	0.006	NKAIN1
		0.961	0.003	NEK4	0.949	0.006	PQLC3
		0.96	0.003	MANSC1	0.949	0.006	RPPH1
5	Kaempferol	**Pa**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.963	0.003	IFIH1	0.96	0.003	GNAI1
		0.961	0.003	COG5	0.96	0.003	LIPH
		0.961	0.003	DNAAF2	0.96	0.003	PITPNM1
		0.961	0.003	FARP1	0.953	0.003	KHNYN
		0.96	0.003	FCHSD2	0.952	0.003	GCKR
		0.96	0.003	KIAA0182	0.953	0.004	CITED2
		0.96	0.003	LGALS8	0.948	0.003	CEP72

		0.96	0.003	MAL2	0.949		
		0.96	0.003	METTL10	0.949	0.006	HSPA12A
		0.96	0.003	MOGAT2	0.949	0.006	LIM2
		0.96	0.003	SATB2	0.949	0.006	MAP3K7IP3
		0.962	0.005	DMXL1	0.949	0.006	NKAIN1
		0.958	0.003	PLSCR4	0.949	0.006	PQLC3
6	catechins,	**Pa**	**Pi**	**DownRegulation**	**Pa**	**Pi**	**UpRegulation**
		0.942	0.005	C6ORF48	0.949	0.004	HIVEP1
		0.94	0.004	DEK	0.931	0.004	RGL1
		0.928	0.004	GSS	0.931	0.004	SLC39A4
		0.92	0.005	TEFM	0.893	0.005	SLC20A1
		0.918	0.007	ITGAV	0.867	0.004	NOTCH1
		0.903	0.008	SLC30A1	0.861	0.031	GFRA1
		0.847	0.006	NPM1	0.831	0.014	GAS6
		0.879	0.042	TBC1D9	0.814	0.041	TMEM38B
		0.845	0.018	EFNB2	0.787	0.02	CTTN
		0.858	0.039	C9ORF41	0.794	0.038	C1ORF63
		0.855	0.038	H1FX	0.791	0.055	BLM
		0.848	0.059	HEATR3	0.784	0.049	CITED2
		0.829	0.041	HIG2	0.773	0.051	CEP72
		0.798	0.014	RASA1	0.755	0.042	ABCC13
		0.82	0.038	YPEL5	0.755	0.042	AURKB
		0.802	0.036	PLEC	0.755	0.042	CLIC6
		0.792	0.04	STEAP1	0.755	0.042	CSRP2BP
		0.772	0.031	PHF14	0.755	0.042	CTPS1

Table 5 will describe the cellular gene expressions that are induced by the chemical compounds. The table will provide an overview of the specific genes that are up or downregulated in response to these chemical compounds when their provability of activity is greater than 0.7 (i.e., Pa>0.7). The genetic influence of a chemical is essential for developing safer and more effective treatments. Thus, this table will be useful in drug discovery.

Table 6 Molecular Docking Analysis with Different Visualization Software (Discovery Studio and Pymol).

S/N	Docking Software	Visualization Software	Protein	Ligand	Binding Affinity (Kcal/mol)	Amino Acid Residue with H-Bond Interaction	Amino Acid Residue with Hydrophobic and other Interaction (B & N are Protein Chains)

1	Auto Dock Vina (vision 1.5.7)	Discovery Studio (vision 20.1.0 19295 copy right 2019)	1G5G (Fragment of fusion protein)	Beta Sitosterol	-8.5	THR219E THR218F THR219D THR219F	GLY222D PRO223D
			1G5G (Fragment of fusion protein)	catechins,	-8.2	THR219D THR219F	PRO223D PRO223E
		PYMOL (vision 3.0.3 copyright© Schrödinger LLC	1G5G (Fragment of fusion protein)	Kaempferol	-7.9	THR219D THR218D GLY222E	PRO223F PRO223D
			1G5G (Fragment of fusion protein)	Morumoside A	-7.7	THR219A THR219B	GLY222B PRO223B
			1G5G (Fragment of fusion protein	Quercetin	-6.3	ARG86C ASN447B	TYR444B LYS446B
			1G5G (Fragment of fusion protein)	Moringine	-4.2	GLY377E THR380E ALA378E	

The table 6 above is for molecular docking Analysis, which provides insights into the binding affinity between a ligand and a target protein. This data will be extremely useful in assessing the potential efficacy of drug candidates. This table can be used to determine the binding affinity scores as well as interactions between the various ligands and target protein (fragment of fusion protein, 1G5G). This information will be essential for predicting compound biological activity, guiding lead molecule optimization, and, ultimately, expediting the development of new drugs. The table's data also may help to validate experimental results and provides an avenue for further in silico studies

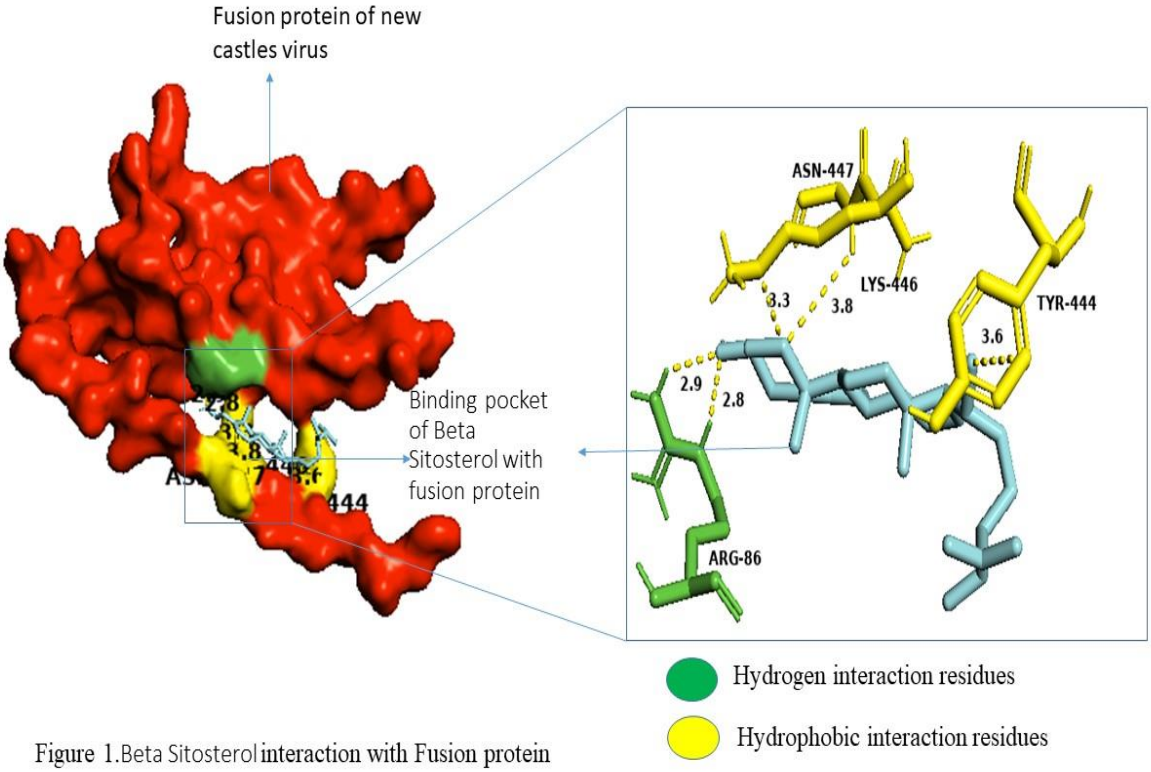

Figure 1.Beta Sitosterol interaction with Fusion protein

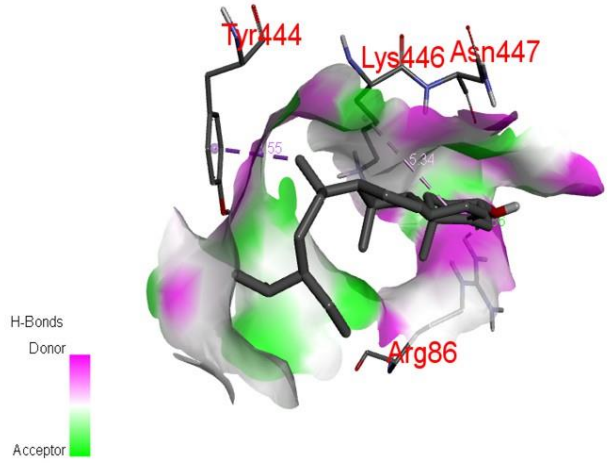

Fig 7A. 3D interactions of Beta Sitosterol with fusion protein

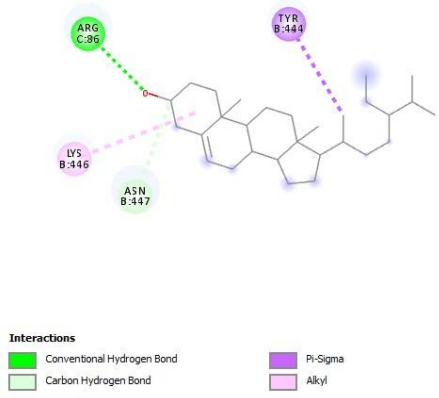

Fig 7B.2D interaction of Beta Sitosterol with F protein

The visualization of binding interaction of fusion protein with Beta Sitosterol by Pymol and biovia discovery studio.

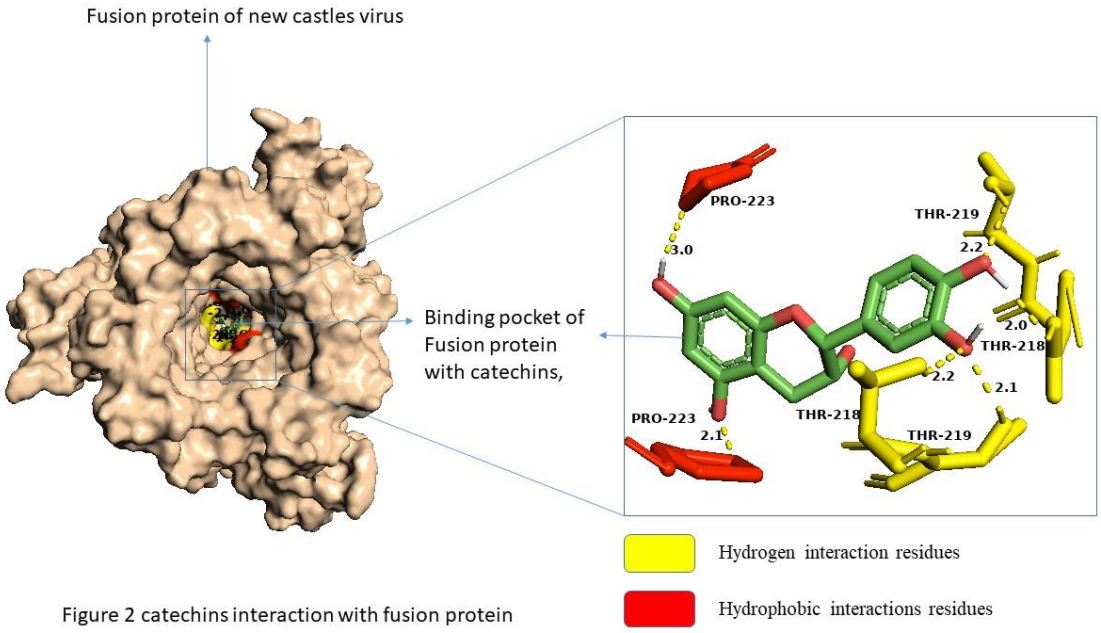

Figure 2 catechins interaction with fusion protein

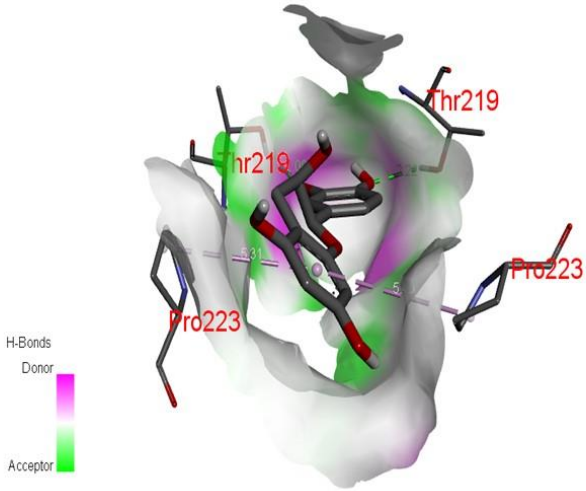

Fig 8A. 3D Catechins interactions with fusion protein

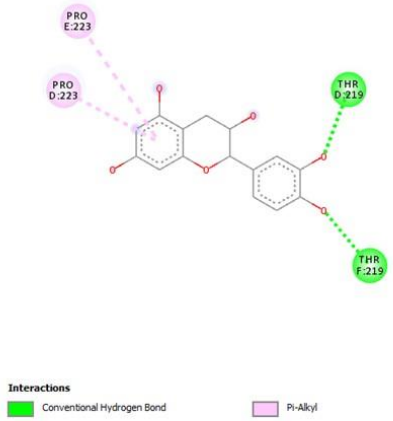

Fig 8B 2D Interactions of catechins with Fusion protein

The visualization of binding interaction of fusion protein with catechins by Pymol and biovia discovery studio

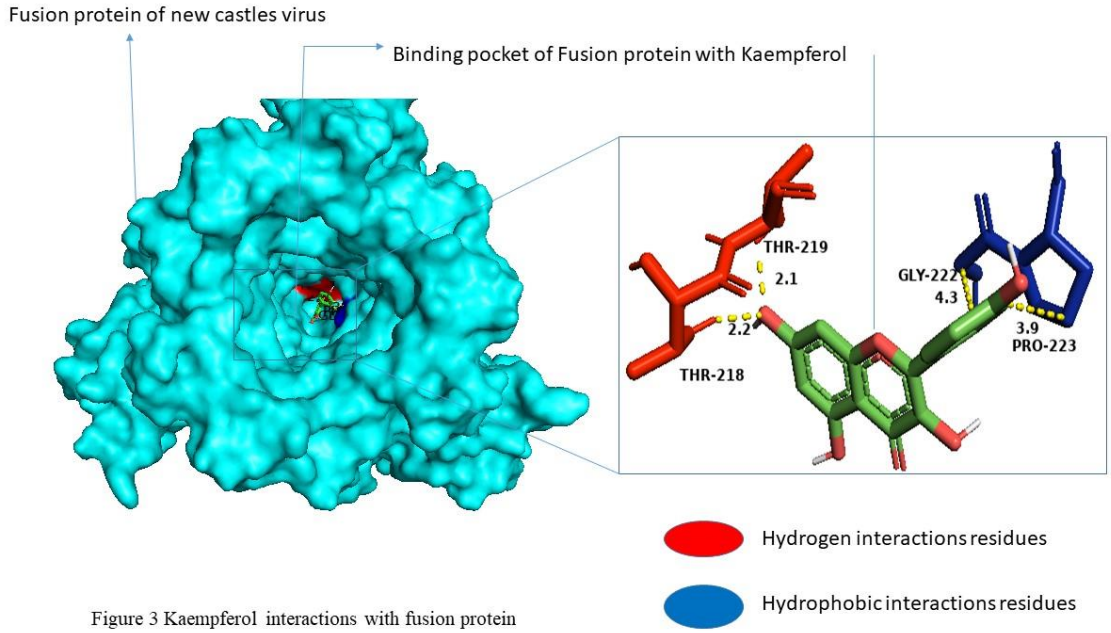

Figure 3 Kaempferol interactions with fusion protein

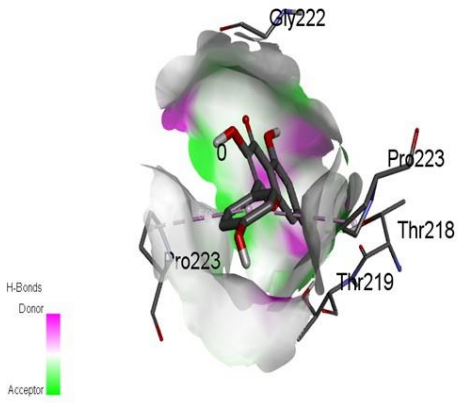

Fig 9A. 3D Interactions of Kaempferol with Fusion protein

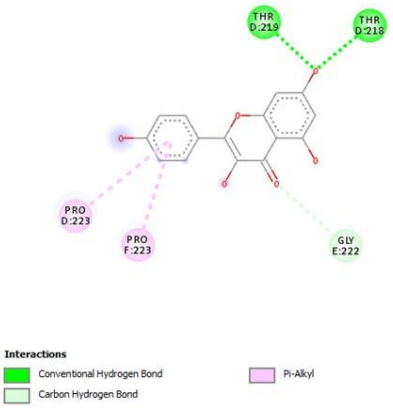

Fig 9B. 2D Interactions of Kaempferol with fusion protein

The visualization of binding interaction of fusion protein with Kaempferol by Pymol and biovia discovery studio

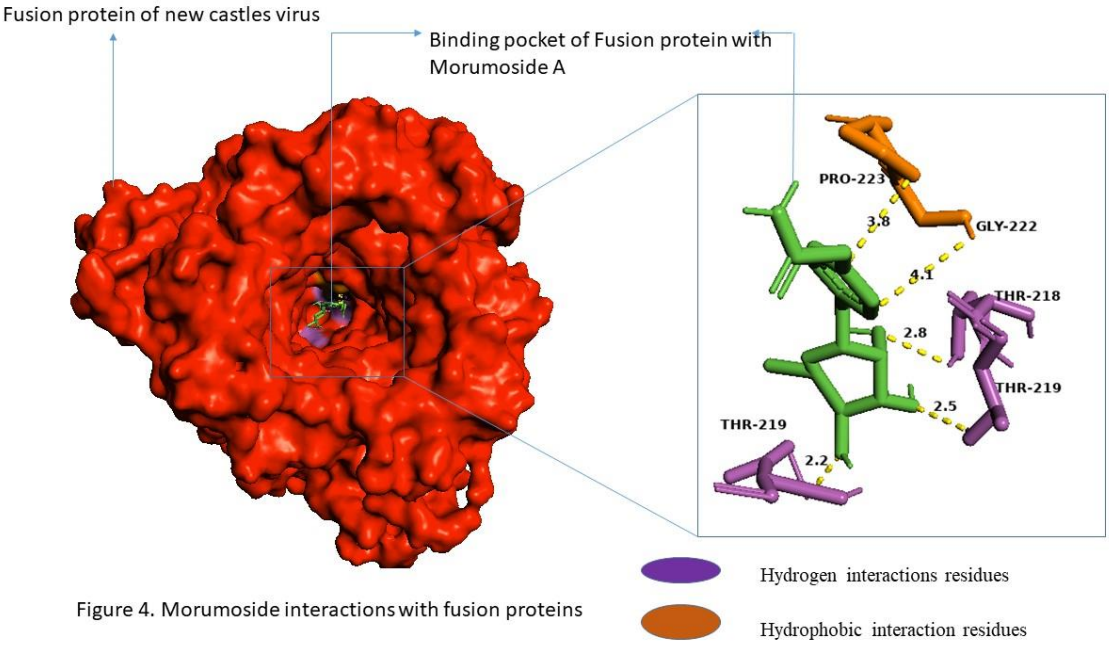

Figure 4. Morumoside interactions with fusion proteins

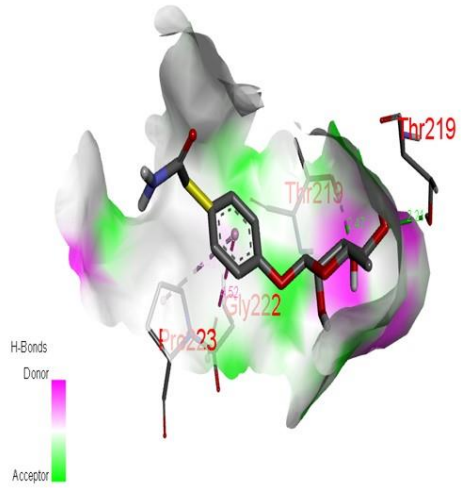

Fig 10A. 3D interactions of Morumoside A with fusion protein

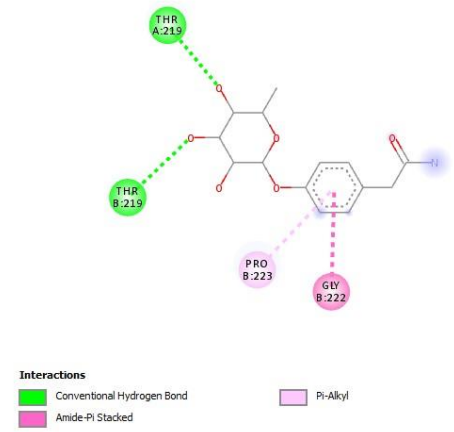

Fig 10B. Interaction of Morumoside A with fusion protein

The visualization of binding interaction of fusion protein with Morumoside A by Pymol and biovia discovery studio

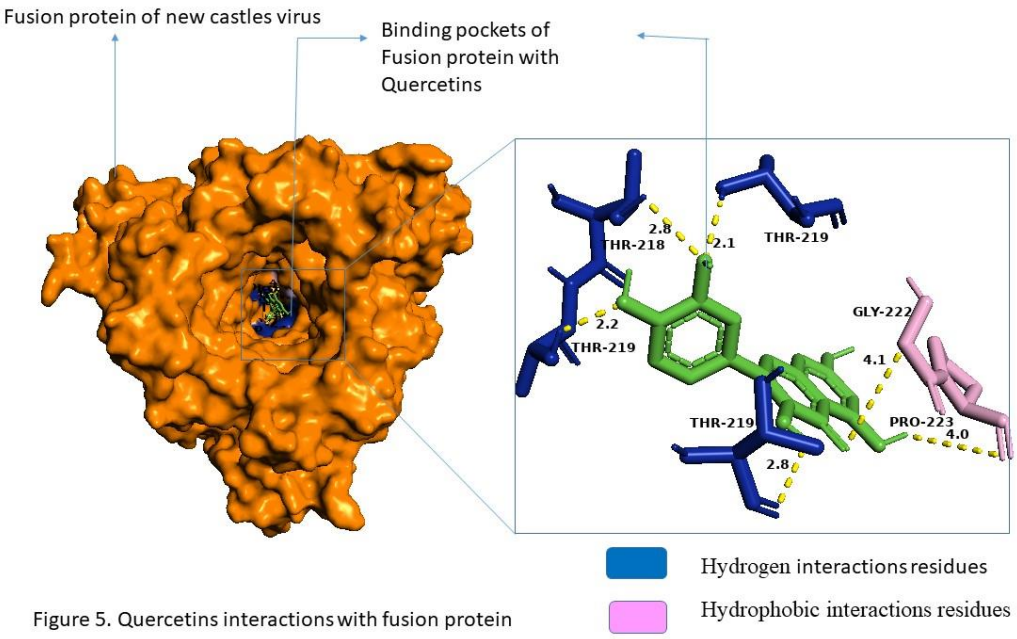

Figure 5. Quercetins interactions with fusion protein

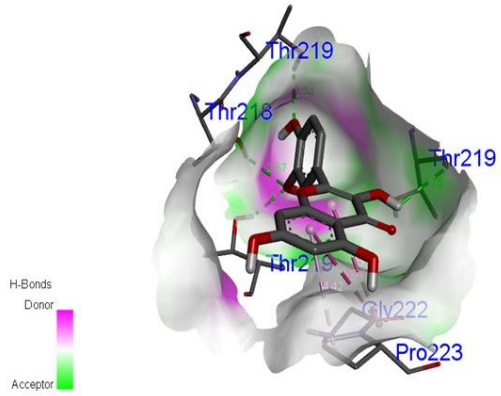

Fig 11A. Interaction of Quercetin with Fusion protein

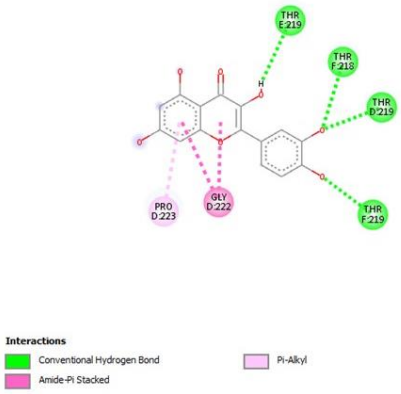

Fig 11B. Interaction of Quercetin with Fusion protein

The visualization of binding interaction of fusion protein with Beta Quercetin by Pymol and biovia discovery studio

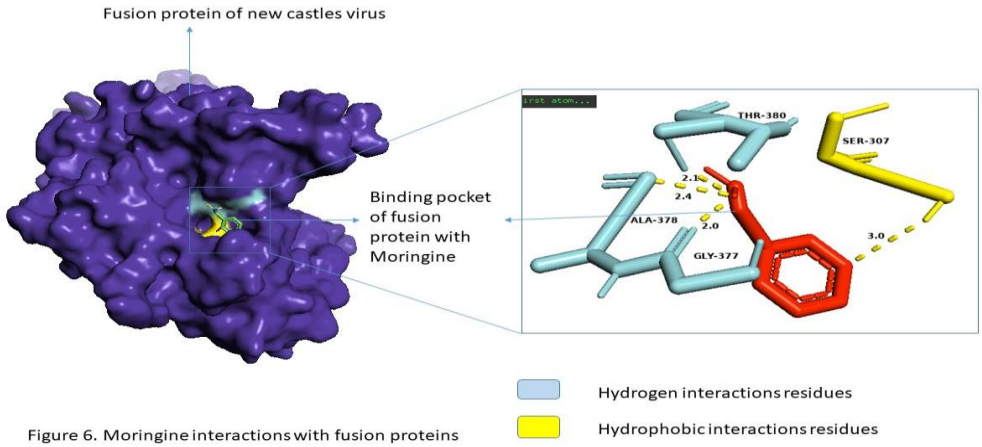

Figure 6. Moringine interactions with fusion proteins

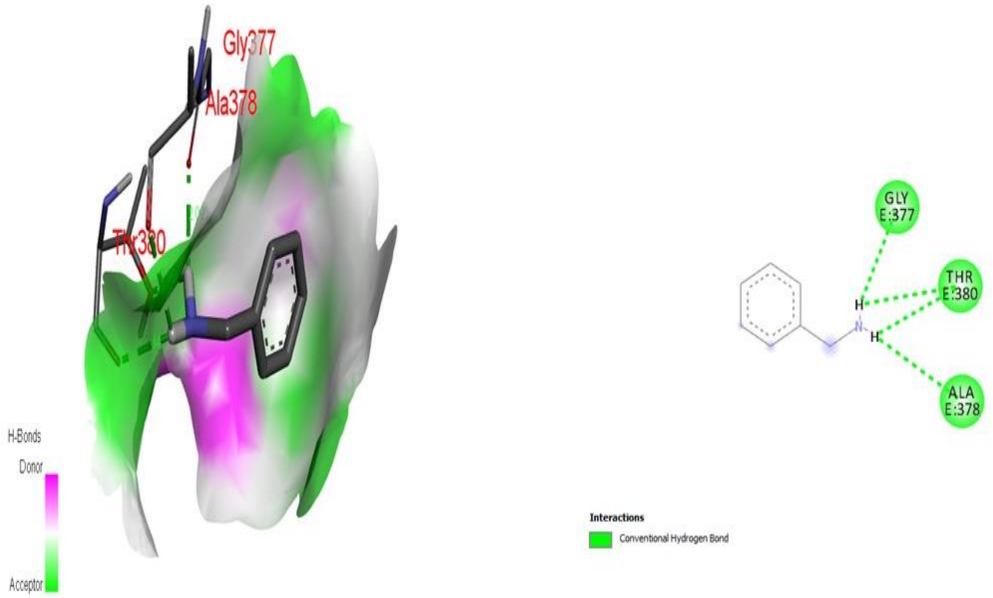

Fig 12A. Interactions of Moringine with Fusion protein Fig 12B interactions of Moringine with Fusion protein

The visualization of binding interaction of fusion protein with Moringine by Pymol and biovia discovery studio

DISCUSSION

Swiss ADME and FAFDrugs4 analysis

Out of 30 phytocompound of Moringa oleifera plants was downloaded from PubChem and was screened using Swiss ADME and FUFDRUGS 4 software to evaluate the potential pharmacokinetics and drugs likeness, based on Lipinski's "Rule of five" a molecules with less reasonable phytocompound properties ware discarded leading to the selection of six (6) with potential drugs likeness properties (molecules weight<500, number of hydrogen donor 5, number of hydrogens acceptor<10, MR Between 30-150. Wlogp3 is <5 except for Beta Sitosterol which is >5 (8) and Xlogp3 is <5 except Beta Sitosterol With >5 (9) [22]. Moreover, bioavailability, Absorption rate, distribution, metabolisms, excretion, toxicity of the 6 molecules was screened 5 out of the six molecules found to have high intestinal absorption and only one have low intestinal absorption (Beta Sitosterol), 5 molecules not crossing blood brain barrier, one will cross blood brain barrier, (Moringine), good water solubility, good oral Bioavailability Both in EGAN and VEBER Rules, as well as traffic light is good, this result is in line with the finding of [23]. but has a slight difference in Xlogp3 and Wlogp3 as showed by one molecule (Beta Sitosterol), as showed in table 1 and figure 13

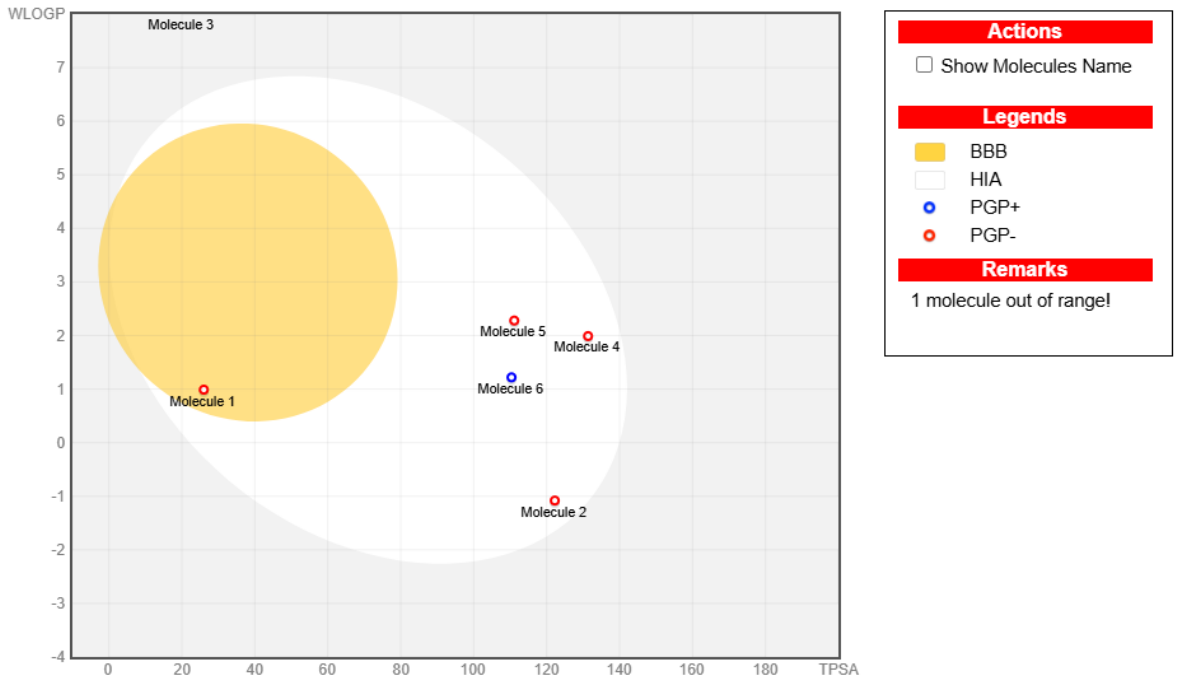

Figure 13 Schematic representation of perceptive evaluation of passive gastrointestinal absorption (HIA) and Brain penetration (BBB) with molecules in the WLOGP-versus-TPSA using BOILED-Egg.

Pass online and adverse and toxic effects software prediction of phytochemicals

Base on the screening of several chemical compounds from Moringa oleifera using pass online and adverse effects software, its predicted biological activities that may be relevant for treating Newcastle Disease Virus (NDV), particularly targeting the Fusion (F) protein of paramyxovirus serotype 1. The biological activities of these compounds, as predicted by software, are likely to contribute to their therapeutic effects, the parameter set in the software during screening is as Pa > Pi and Pa > 0.7.17 of the six total MO chemicals. Moringine shows a high probability of inhibiting various enzymes, such as Complement factor D, Polyamine-transporting ATPase, and Glucose oxidase. These activities could potentially interfere with the viral life cycle, reduce inflammation, or enhance the host's immune response whereas, Morumoside A Predicted to act as a Vasoprotector and Membrane integrity agonist, which could protect the cells from the cytopathic effects of the virus, maintaining cellular integrity during infection, Beta Sitosterol Shows strong inhibition of Prostaglandin-E2 9-reductase and Cholesterol antagonist activity, This could reduce inflammation and modulate lipid pathways, potentially affecting viral replication by altering the host cell environment, while Quercetin was Predicted to inhibit HIF1A expression and Peroxidase activity, while enhancing Membrane integrity. This suggests a role in reducing oxidative stress and stabilizing cellular membranes, possibly limiting virus-induced cell damage, and Kaempferol: Acts as a Membrane integrity agonist and HIF1A expression inhibitor, similar to quercetin, with additional activities like inhibition of Chlordecone reductase and Antihemorrhagic properties, which might help in reducing viral replication and protecting the host cells. Moreover the phytocompound showed some adverse effects when used as drugs by the target animals as Moringine: Could cause metabolic acidosis, pure red cell aplasia, and multiple organ failure at higher doses.

However the phytocompound from MO shows some adverse effects after screening as indicate Morumoside A, Potential to cause hypercholesterolemia, inflammation, and hematotoxic effects, whereas Beta Sitosterol Might lead to sleep disturbances, reproductive dysfunction, and ocular toxicity, Quercetin is Associated with vascular toxicity, shivering, and reproductive dysfunction, Kaempferol Genotoxic and might cause vascular toxicity, shivering, and inflammation and Catechins: Could lead to inflammation, vascular toxicity, and neurotoxicity [24] as show in table 2 and 3

SEA Search Server software prediction

The six (6) MO phytochemicals' P-Value, MaxTC, Target Name, Target Key, Query Molecule, and Description were displayed in Table 4.4. Using structural similarity to pharmaceuticals as a basis for prediction of binding affinity, compounds that are predicted to bind to target proteins were found using the Similarity Ensemble Approach (SEA) search server software. As demonstrated, for instance, in the table, these substances are anticipated to have substantial binding affinities with various human, viral, and animal proteins. Moringine Among other proteins, it was discovered to interact with Lysyl oxidase homolog 2 (LOXL2). The extracellular matrix's cross-linking of collagen and elastin, which is related to LOXL2, may be important for immunological response and tissue repair. Because of its interaction with this protein, Moringine may be able to regulate immunological responses, which could be advantageous.
Morumoside A chemical was found to interact with the sodium/glucose cotransporter 2 (Slc5a2). Slc5a2 regulates glucose uptake in cells, which may influence viral replication because viruses frequently rely on the host's metabolic processes for replication. By blocking Slc5a2, Morumoside A may impair the virus's ability to replicate.
Beta Sitosterol. It had a high binding affinity for the Vitamin D3 receptor (VDR) as well as enzymes involved in steroid metabolism. The Vitamin D3 receptor is known to influence the immune system, and Beta Sitosterol may boost the host's immunological response to NDV by interacting with VDR, making it more difficult for the virus to develop an infection. It was discovered that the flavonoid quercetin interacts with the enzyme arachidonate 15-lipoxygenase (ALOX15), which is involved in the regulation of inflammation and the metabolism of fatty acids. Quercetin may lessen the inflammation brought on by an NDV infection, which is a significant contributing factor to the severity of the illness, by blocking ALOX15. Kaempferol It interacted with Fatty acid synthase (FAS), an important enzyme in lipid production. Inhibiting FAS may lower the supply of lipids that the virus requires for reproduction, decreasing the virus's capacity to spread. This result is same with the [24] as indicated in table 4.

DIGEP Pred software prediction

The parameters of probability of bioactivity were set as Pa > Pi and Pa > 0.7.17 during DIGEP Pred screening. The total of six (6) phytocompound of Moringa oleifera was screened using DIGEP-Pred software and found to have good biological activity which is related to up and downregulation to the targets protein (Fusion protein (1G5G) and other human and animals protein The expression of gene by the MO phytocompound analyzed using DIGEP-Pred, Compounds have been shown to positively or negatively affect genes involved in diverse biological activities. In our investigation, we chose some genes. IFIT1, GNAI1, IFIH1, CHRNE, CACNG4, MAST4, TCF12, MAPK8, PPARG, FASN, NQO1, and IL6. This expressed genes play a direct role in the virus's immunogenicity and cytokine modulation during disease. Genes that are upregulated by Kaempferol, such as GNAI1 (Guanine nucleotide-binding protein G subunit alpha-1), may play a role in signalling pathways that enhance the immune response against the

virus, potentially reducing the effectiveness of NDV's fusion protein in infecting host cells and Downregulation of genes like IFIH1 (Interferon-induced helicase C domain-containing protein 1) might affect the bird's innate immune response, potentially making it more susceptible to NDV if these antiviral mechanisms are weakened, The upregulation of genes like CACNG4 and CHRNE by beta-Sitosterol may enhance certain cellular pathways that could strengthen the bird's immune response or cellular defence mechanisms, potentially making it harder for NDV to replicate or spread, WISP2 is involved in the regulation of cellular growth and may contribute to tissue repair and immune responses, upregulating antiviral genes like IFIT1 by Moringine could help the birds' immune system combat the virus more effectively. The upregulation of genes like TCF12 and SMPD1 by Morumoside A may influence pathways related to immune response and cellular signalling. For example, SMPD1 is involved in the metabolism of sphingomyelin, which can play a role in cell membrane integrity and signalling, potentially affecting how cells respond to viral infections, and the downregulation of genes such as MAPK8 and ADAM19 Quercetin might alter the bird's immune response or other cellular processes. MAPK8, for instance, is part of the MAP kinase pathway, which is crucial for signalling related to stress and immune responses. Its downregulation could potentially weaken the bird's ability to respond to viral infections like NDV, The upregulation of genes like PPARG and ELOVL6 could enhance certain metabolic pathways and immune responses. PPARG, for instance, plays a role in regulating fatty acid storage and glucose metabolism, which might influence the overall energy balance and immune function of the bird, potentially aiding in the fight against viral infections like NDV, The downregulation of genes such as FASN and SREBF1, Quercetin which are involved in lipid biosynthesis and metabolism, might reduce the availability of resources that viruses like NDV need for replication. This could potentially hinder the virus's ability to replicate effectively within the host, and finally, the upregulation of genes like NQO1 and SOD1, catechins which are involved in antioxidant defence, can help protect cells from oxidative stress caused by viral infections like NDV. This can enhance the bird’s ability to cope with the viral attack and reduce cellular damage, potentially limiting the severity of the infection, downregulating pro-inflammatory genes like IL6 and TNF, catechins might reduce the inflammation caused by NDV. While inflammation is part of the immune response, excessive inflammation can be harmful and contribute to tissue damage during infection. Catechins might help mitigate these negative effects, leading to a more controlled immune response. The result is line with finding of [24] as showed in the table 5

Protein-Ligand interaction analysis

The ligand-protein interaction are generally highlighted in hydrogen and hydrophobic bonding, which play an important role in the prediction of binding affinity of ligand to protein, all selected six ligand or phytocompound ware docked with SR1 of fragment of fusion protein (1G5G) in the active and sialic acid site, fig 1-12 (both Pymol and discovery studio) illustrate the binding interaction of the selected natural chemicals in the binding pocket of the target protein The active site of the target protein showed hydrogen and hydrophobic interactions with various amino acid of the protein which include THR218, THR219, THR380, GLY222, GLY377, ARG86, ASN447, ALA378 and GLY222, PRO223, TYR444, and LYS446 respectively.

The foremost binding interaction of the amino acid residue are Beta Sitosterol (-8.5kcal/mol) showed two H bond with amino acid THR218F, THR219E,THR219D,THR219F and hydrophobic bond with amino acid GLY222D and PRO223D, whereas catechins molecule with score of (8.2 kcal/mol) showed one H bond with amino acid THR219D,THR219F and hydrophobic bond with PRO223D,PRO223E and that of

Kaempferol (score -7.9 kcal/mol) with three H bond of THR218D, THR219D, GLY222E and also with one hydrophobic bond of PRO223F, and PRO223D Moreover Morumoside A with (score -7.7 kcal/mol) showed one H bond with THR219A, THR219B and two hydrophobic bond with GLY222B and PRO223B, whereas, Quercetin with score of (-6.3 kcal/mol) formed a two H interaction with ARG86C, and ANS447B and hydrophobic interaction with amino acid residue of TYR444B and LYS446B, finally Moringine (-4.2 kcal/mol) displayed only three H interaction with amino acid residue of GLY377E, THR380E and ALA378E respectively which slightly differs with find of [25]. In term amino acid residue. As showed in table 6

Conclusion

Conclusively the study showed Moringa oleifera phytochemicals have good novel inhibitory activity against the target protein fragment of fusion protein (paramyxovirus serotype 1) (APMV-1) of new castles diseases of birds especially chicken as is the most predominant birds species that usually affected by the diseases, however the finding pointed out several compounds which are suitable for molecular docking with pharmacokinetic analysis and biological activity predictions, such as Quercetin, Morumoside A, Moringine, but Kaempferol, catechins and Beta Sitosterol in the finding serve as promising chemical for treatment of the diseases but displayed high lipophilicity and water solubility which is greater than 5 [which is >5 (8) and Xlogp3 is <5 except Beta Sitosterol With >5 (9)], but the other five phytochemicals have an optimal lipophilicity and water solubility which make them suitable for taking them as good candidate for drug against new castle diseases. Moreover, Moringine have showed the problems of blood-brain barrier penetration, their diverse biological activities, including antioxidant, anti-inflammatory, and anticancer characteristics, highlight their potential therapeutic applications beyond New castle diseases treatment. This thorough in silico analysis supports the advancement of Moringa oleifera phytochemicals as promising candidates facilitating the development of new, safer, and more effective new castle diseases of birds treatments, potentially decreasing reliance on high costly synthetic drugs and their accompanying side effects

Furthermore, a research should also be conducted to further more findings on some phytochemicals that can be as good candidate for the treatment of new castle diseases as it has no specific drugs for treatment

Author contributions:

RD: conceptualization, Data curation, Data analysis, Manuscript Editing, **UAA**: Data curation, Data Analysis, Manuscript draft writing and editing, **SSM:** Data analysis, Manuscript editing, Manuscript editing and proof reading**, IAG**: Data analysis, Manuscript editing, Manuscript editing and proof reading, **ARA**: Review and Manuscript editing.

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