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Unveiling the Anticancer Efficacy of Kaempferol from *Moringa oleifera* Against Breast Cancer: Insights from an In Silico Investigation

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Abstract
Breast cancer is a significant global health problem that is often encountered in women. Kaempferol from *Moringa* is known to control breast cancer cells. This study explores the in silico potential of kaempferol compounds derived from *Moringa oleifera* as breast cancer drug candidates. The results showed that kaempferol compounds have anticarcinogenic potential, with a high Pa value of 0.715, via the target proteins CYP1B1 and HSD17B2. The prediction of physicochemical properties indicates that kaempferol is a viable oral medication candidate, as it adheres to *Lipinski's rule of five*. However, it is dangerous if swallowed because it is classified as toxicity class 5. Molecular docking analysis indicates that kaempferol has high affinity for the target proteins CYP1B1 and HSD17B2. It can significantly contribute to breast cancer progression, with binding energies of -7.1 kcal/mol and -6.8 kcal/mol, respectively. Hydrogen bonds and hydrophobic interactions predominated the molecular interactions between kaempferol and the target proteins. These findings suggest that kaempferol from *Moringa oleifera* is a promising lead compound for the development of breast cancer treatments. Despite its potential, further studies are required to mitigate its toxicity risks, as indicated by the pharmacokinetics, drug-likeness, and toxicity assessments.

Keywords: Breast cancer; Kaempferol; *Moringa oleifera*

1. INTRODUCTION

Cancer is a condition characterized by unregulated cellular proliferation that may metastasize to other regions of the body (Saini *et al*., 2020). Breast cancer is the most prevalent malignancy identified in women (Harbeck *et al*., 2019). Breast cancer ranks as one of the foremost causes of mortality among women globally, accounting for one-quarter of cancer diagnoses and one-sixth of cancer fatalities (Luo *et al*., 2022). Breast cancer (carcinoma mammae) is a malignancy originating from the cells of breast tissue, namely the ductal epithelium and lobules. Breast cancer arises from cells that have lost their normal regulatory mechanisms, leading to aberrant, rapid, and uncontrolled proliferation (Sun *et al*., 2017). In the treatment process, breast cancer can be treated with various treatments, one of which is chemotherapy. Chemotherapy uses chemicals to inhibit or kill cancer cells. However, this treatment has severe side effects and is costly (Saini *et al*., 2020). Therefore, alternative treatments are needed, including the use of potential compounds derived from natural materials.

Moringa oleifera L. has been reported to possess anticancer activity against breast cancer, largely attributed to its flavonoid constituents. Experimental studies have demonstrated that *Moringa oleifera* extracts can suppress breast cancer cell proliferation, reduce cell motility, and inhibit tumor formation by modulating cancer-related signaling pathways (Al-Asmari et al., 2015). Among its bioactive compounds, kaempferol has attracted attention due to its reported ability to inhibit cancer cell growth, suppress angiogenesis, and induce apoptosis (Edwinanto et al., 2018).

Moringa oleifera L. has been reported to exhibit anticancer activity against breast cancer, which has been attributed to its multiple flavonoid constituents (de Morais et al., 2024). Among these bioactive compounds, kaempferol has been widely studied due to its reported anticancer-related biological and molecular activities, particularly in breast cancer models. Experimental studies have demonstrated that kaempferol inhibits breast cancer cell proliferation, induces G2/M phase cell cycle arrest, and triggers apoptosis by activating caspases in MDA-MB-231 cells (Kaur et al., 2025). In addition, kaempferol suppresses migration and invasion in triple-negative breast cancer models by downregulating RhoA and Rac1 signaling pathways and modulating key regulators of cell division such as CDKs and PD-L1 (Al-Asmari et al., 2015). While other flavonoids present in *M. oleifera* may also contribute, individually or synergistically, to its anticancer effects, these findings support selecting kaempferol as a representative flavonoid for further mechanistic investigation beyond its general antioxidant properties.

Kaempferol is a widely distributed flavonoid aglycone, commonly found in plants as glycosides. Structurally, kaempferol is a tetrahydroxyflavone with hydroxyl groups at positions 3, 5, 7, and 4′. Previous studies have shown that kaempferol modulates breast cancer cell behavior through multiple mechanisms, including regulation of cellular redox balance by suppressing the NF-κB pathway and activating the Nrf2 transcriptional pathway (Imran et al., 2019).

Scientific and technological developments have enabled drug research and development using computational methods, also known as in silico studies. Prior to in vitro and in vivo experiments, in silico methods enable early assessment of bioactive compounds, offering time- and cost-saving benefits (Effendi, 2023). Previous in silico studies have explored the pharmacological potential of kaempferol, derived from *Moringa oleifera* or other plant sources, primarily through molecular docking and ADME prediction against various cancer-related targets. However, these studies have largely focused on general anticancer activity or interactions with isolated molecular targets, without specifically addressing hormone-dependent mechanisms relevant to breast cancer. Moreover, there are still a few thorough computational methods that combine molecular docking, pharmacokinetic profiling, toxicity evaluation, and target protein prediction into a single framework. These gaps show that a more comprehensive in silico study is required to understand better the possible molecular mechanisms of kaempferol in breast cancer.

2. MATERIAL AND METHODS

2.1. Structure preparation and analysis of natural compounds as potential drugs

The 3D structure of kaempferol (PubChem CID: 5280863) was retrieved from the PubChem database (https://www.ncbi.nlm.nih.gov/) in PDB format. The compound was prepared by energy minimization and converted into the appropriate format for subsequent analyses. Prediction of biological activity was conducted using the PASS (Prediction of Activity Spectra for Substances) online server via the Way2Drug platform (https://www.way2drug.com/passonline/). The analysis focused specifically on anticarcinogenic activity to support the evaluation of kaempferol's potential anticancer properties based on predicted biological functions rather than antioxidant activity alone (Druzhilovskiy et al., 2017).

2.2 Analysis of pharmacochemical properties and toxicity test of kaempferol compounds

The drug-likeness and pharmacokinetic properties of kaempferol were evaluated using the SwissADME webserver (http://www.swissadme.ch/). Parameters assessed included molecular weight, lipophilicity (MLOGP), hydrogen bond donors and acceptors, and molar refractivity, following *Lipinski's rule of five* to estimate oral bioavailability (Jadhav *et al*. 2015). Toxicity prediction was performed using the ProTox-II webserver (https://tox.charite.de/protox3/) to estimate the median lethal dose (LD50) and toxicity class, providing an early assessment of safety risks relevant to anticancer drug development (Banerjee et al., 2018).

2.3. Target protein identification and visualization

Potential protein targets of kaempferol were predicted using the SuperPred webserver (https://prediction.charite.de/) using the canonical SMILES structure. The predicted targets were subsequently analyzed using the STRING database (https://string-db.org/) to construct protein–protein interaction (PPI) networks and to identify significantly enriched biological pathways through KEGG pathway analysis. The STRING–KEGG analysis revealed that several predicted targets were involved in hormone metabolism, steroid biosynthesis, and estrogen-related signaling pathways, which are critically implicated in breast cancer development and progression. Prior to docking, the structural quality of the selected protein models was validated using standard parameters, including resolution, Ramachandran plot statistics, and the absence of significant structural abnormalities, to ensure the reliability of the docking simulations.

2.4. Molecular docking and visualization of docking results

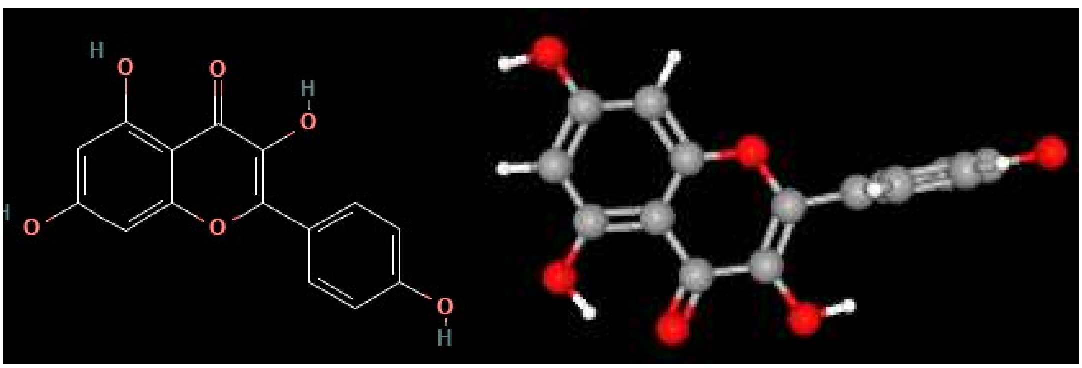
Molecular docking between ligand and target protein using PyRx application and analyzed with AutoDock Vina program to determine the binding affinity value (Kusuma et al., 2022). In addition, docking aims to achieve optimal protein and ligand conformation (Setiawan & Irawan, 2017). The interaction results were visualized as a 2D structure using the Protein Plus webserver (https://proteins.plus/) and as a 3D structure using the PyMOL application.

3. RESULTS AND DISCUSSION

3.1. Structure and Potential of Kaempferol as Drugs

Information on the active compound kaempferol was obtained from the PubChem database at NCBI. The database structure shows that kaempferol is a flavonoid commonly found in plants, including *Moringa* (Figure 1). Other data obtained from the PubChem web database are the Canonical SMILES of kaempferol compounds, namely C1=CC(=CC=C1C2=C(C(=O)C3=C(C=C(C=C3O2)O)O)O)O.

The results show that kaempferol belongs to the flavonoid group. This is substantiated by its structure, which features a 15-carbon benopyranone or benzopyran, in which the three-carbon bridge between the phenyl groups is often cyclized with oxygen, resulting in a C6-C3-C6 flavan nucleus (Periferakis et al., 2022). Kaempferol, one of the main flavonols in *Moringa oleifera* L. leaves, has potential as an anticancer agent for breast cancer treatment. This is because kaempferol can inhibit cell growth, induce cell cycle arrest in the G_2/M phase, and increase the expression of apoptotic proteins such as cleaved caspase-3 and γH2AX, indicating an anticancer mechanism through apoptosis and DNA damage (Zhu and Xue, 2019).



(a) (b)

Figure 1. Structure (a) 2D and (b) 3D of the kaempferol compound.

The data were used to test drug potency using the Way2Drug online PASS webserver (Table 1). The analysis showed that kaempferol can be used as an anticarcinogenic drug. The potential bioactivity of kaempferol was first investigated using PASS, which yielded a probable activity (Pa) value of 0.715 (Table 1). This Pa score is a computational prediction of anticarcinogenic action and does not provide direct evidence of biological effectiveness. As a result, the PASS results were only used as a preliminary screening method, and they were further analyzed by combining them with predicted molecular targets and previously reported experimental data on kaempferol's anticancer activity. In particular, predicted activity was consistent with molecular docking analyses against breast cancer-related targets and supported by existing in vitro evidence of kaempferol's effects on cancer cell proliferation, death, and migration. According to Prasetyorini *et al.* (2022), a Pa > 0.7 indicates that the molecule has substantial computational potential and may be used as a drug at the laboratory scale. Kusuma *et al.* (2022) further showed that a higher Pa value correlates with a greater likelihood of the substance blocking receptors in laboratory trials.

Table 1. Medicinal potential of kaempferol.

Compound	Potential	Pa	Pi
Kaempferol	Anticarcinogenic	0,715	0,008

The potential bioactivity of kaempferol was initially estimated based on the predicted probability of activity (Pa) value of 0.715 obtained from the PASS analysis (Table 1). Accordingly, the PASS result was used solely as a preliminary screening tool to support subsequent molecular analyses rather than as a standalone indicator of drug potency. The predicted anticarcinogenic potential of kaempferol is further supported by target protein prediction and molecular docking analyses, which identified CYP1B1 and HSD17B2—key enzymes involved in estrogen metabolism and breast cancer progression—as plausible molecular targets. Moreover, previous experimental studies have demonstrated that kaempferol inhibits proliferation, induces apoptosis, and causes cell cycle arrest in breast cancer cell lines, thereby providing biological relevance to the computational predictions (Yi et al., 2018; Imran et al., 2019; Al-Asmari et al., 2015).

3.2. Physicochemical properties and toxicity of kaempferol

Analyzing the physicochemical properties of a compound as a drug candidate is important for determining the likelihood that it can become an oral drug or behave like one (Table 2). The parameters considered can follow *Lipinski's rule of five.* It shows that the molecular weight of kaempferol is well distributed at 286.24 g/mol. This is because molecular weight is closely related to the distribution of compounds. The MLOGP value for kaempferol falls within the specified parameter range of -0.03. The molar refractivity values of kaempferol compounds have also been assessed to meet *Lipinski's rule of five*, with values ranging from 76.01.

Toxicity test analysis is also important for assessing the risk a toxic chemical may pose to humans (Figure 2). Kaempferol is classified as toxicity class 5 under the Globally Harmonized System ($2000 < LD_{50} \leq 5000$ mg/kg) based on its estimated LD_{50} value of 3919 mg/kg. This level indicates relatively low acute oral toxicity and is widely considered a promising safety profile in early drug development. However, it is important to note that this classification is based on in silico prediction and does not account for dose-dependent, chronic, or organ-specific harmful effects. Thus, while the predicted toxicity profile of kaempferol provides a reasonable safety margin for further exploration, experimental toxicological validation is still required to confirm its safety.

Table 2. Physicochemical properties of kaempferol.

Parameters	Value
Molecular Weight	286.24 g/mol
MLOGP	-0.03
Hydrogen Bond Donors (HBD)	4
Hydrogen Bond Acceptor (HBA)	6
Molar Refractivity	76.01

Figure 2. Toxicity prediction results of kaempferol.

3.3 Target Protein Identification and Visualization

The target protein prediction results showed several proteins that can work with kaempferol compounds (Table 3). The target protein are CA7, CYP1B1, CISD1, CA12, HSD17B2, and DPP4. The proteins targeted by kaempferol compounds have different functions in the human body, namely: CA7 and CA12, which regulate pH and ions; CYP1B1 and HSD17B2, which function in hormone and steroid metabolism; CISD1, for mitochondrial homeostasis and iron-sulfur; and DPP4, for glucose and peptide metabolism. These proteins can interact with kaempferol because it has antioxidant, anticancer, anti-inflammatory, and antidiabetic activities.

Two predicted target proteins, CYP1B1 (Q16678) and HSD17B2 (P37059), are involved in estrogen metabolism and have been associated with breast cancer development (Figure 3). In this study, several proteins were identified using in silico target prediction, indicating potential molecular interactions between kaempferol and estrogen-related pathways. Importantly, the current in silico investigation did not show direct biological effects, such as cell growth inhibition or apoptosis induction. Previous research has shown that kaempferol has anticancer properties in breast cancer models, including suppression of cell proliferation, induction of apoptosis, and G2/M phase cell cycle arrest via modulation of the estrogen-related and PI3K/Akt signaling pathways (Imran et al., 2019). These experimental findings confirm the biological relevance of the predicted targets identified in this investigation. However, they should be considered supporting literature rather than direct results of the current computational analysis.

Table 3. Predicted target proteins of kaempferol related to breast cancer.

Protein Target	UniProt ID
CA7	P43166
CYP1B1	Q16678
CISD1	Q9NZ45
CA12	O43570
HSD17B2	P37059
DPP4	P27487

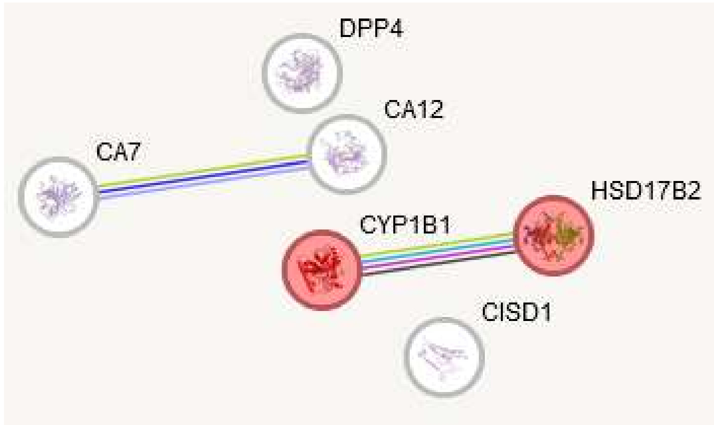
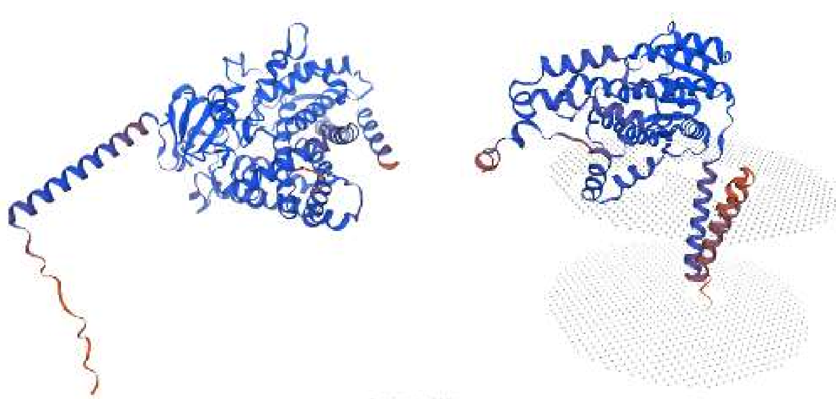


Figure 3. Protein-protein interaction network of predicted target proteins.

The identified target proteins were modeled using the SWISS-MODEL web server (Figure 4). Structure validation was performed through the SAVES web server (Table 4) to determine the quality of the protein structure model and the number of residue plots. The results show that the CYP1B1 protein structure model quality is 95.2741, and HSD17B2 has a value of 99.2021. Validation of the target protein structure is essential before performing molecular docking. It aims to obtain a good protein structure that binds to the ligand. A good protein structure has a high structural quality. A high structural model quality value indicates good quality, as higher values indicate better results. Protein residues should also be considered in structure validation. This is because protein residues will ensure the stability, function, and interactions necessary for its biological role. In addition, it prevents problems such as malfunctions or diseases due to incorrect protein folding. According to Aghnitory et al (2022), a good residue plot is less than 0.8%. The results show that the residue values for protein CYP1B1 and HSD17B2 are also 0.2% and 0.3%, respectively, yielding a good number of residue plots.



(a) (b)

Figure 4. Protein modeling of breasr cancer target proteins. *Description*: (a) CYP1B1 and (b) HSD17B2.

3.4 Molecular docking ligand and protein target

The identified and visualized target proteins were docked and compared with other ligands (drugs) that are commonly used for breast cancer treatment. This molecular docking and comparison step was carried out to determine the binding affinity (Table 5) and the interaction between the protein and the ligand (Table 6). In molecular docking, binding affinity is required to determine the energy required for a compound to bind to the target protein. The smaller the value, the stronger the bond, meaning the compound requires less energy to bind to the target protein.

Table 4. Validation results of target protein models.

Protein Target	Quality	Residue
CYP1B1	95.2741	0,2%
HSD17B2	99.2021	0,3%

Tamoxifen was chosen as a reference ligand because of its role as a selective estrogen receptor modulator (SERM) that is commonly used in the treatment of estrogen receptor-positive breast cancer. Tamoxifen's anticancer effects are principally mediated by estrogen receptor antagonism, which inhibits estrogen-dependent transcription and cell proliferation (Harbeck et al., 2019). In contrast, kaempferol does not act as a SERM. However, it is thought to have anticancer properties by modulating estrogen-metabolizing enzymes such as CYP1B1 and HSD17B2, as well as other cancer-related signaling pathways. As a result, tamoxifen served as a mechanistic reference rather than a standard for therapeutic equivalence, enabling comparisons of binding activity within estrogen-related pathways without assuming clinical equivalence.

Table 5. Binding affinity values of kaempferol and reference ligand against target protein.

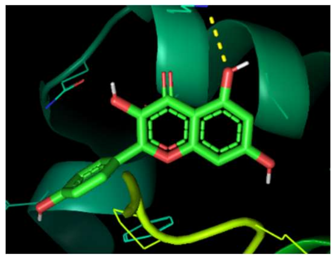
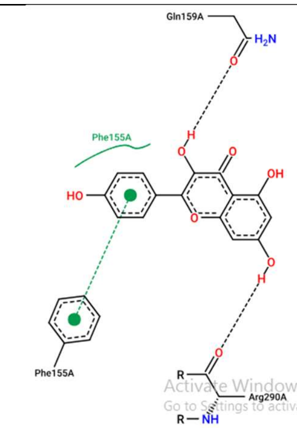
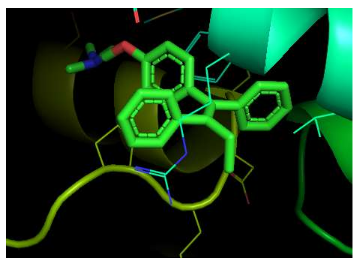
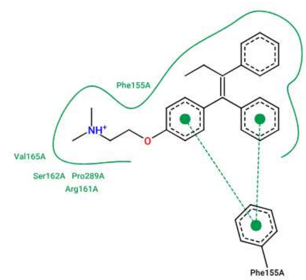
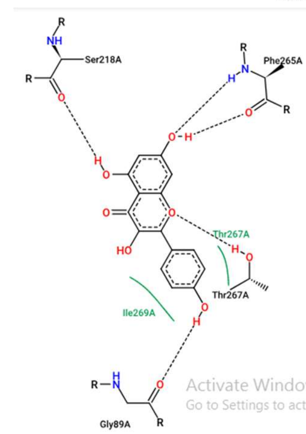
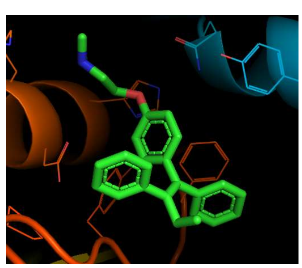
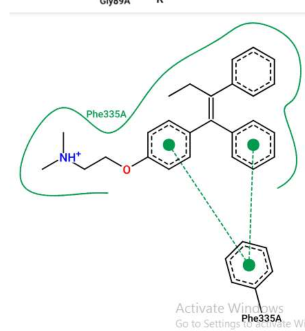
Protein Target	Ligand	
	Kaempferol	Tamoxifen
CYP1B1	-7.1 kcal/mol	-7.6 kcal/mol
HSD17B2	-6.8 kcal/mol	-6.9 kcal/mol

The results show that the binding affinity of CYP1B1 with tamoxifen is higher at -7.6 kcal/mol than with kaempferol at -7.1 kcal/mol. The binding affinity of the HSD17B2 protein with tamoxifen is also higher at -6.9 kcal/mol than that with kaempferol at -6.8 kcal/mol. The binding affinity results indicate that tamoxifen has a higher affinity than kaempferol, so it does not require much energy to bind to the target protein.

Estrogen and progesterone are essential hormones in the human body, significantly influencing breast tissue growth and development. These hormones collaborate to prepare the breast for gestation, facilitating the proliferation and differentiation of mammary gland cells. An imbalance in their levels may facilitate the onset and progression of breast cancer.

Specifically, two key proteins, CYP1B1 and HSD17B2, which are involved in steroid hormone metabolism, have been linked to breast cancer when their activities are dysregulated.

Table 6. Molecular docking visualization of CYP1B1 and HSD17B2 proteins with kaempferol and tamoxifen.

Protein Target	Ligand	Visualization Results	
		3D	2D
CYP1B1	Kaempferol		
	Tamoxifen		
HSD17B2	Kaempferol		
	Tamoxifen		

CYP1B1, part of the cytochrome P450 family, is involved in the oxidative metabolism of various xenobiotics, including carcinogens. Elevated estrogen levels can overstimulate CYP1B1 activity, leading to metabolic signals that promote the growth of breast cancer cells. Conversely, HSD17B2 regulates estrogen levels by converting estradiol, a potent estrogen, into estrone, a weaker form. Dysregulation in this conversion can result in higher estradiol levels, thereby enhancing the proliferation of breast cancer cells. Thus, the balance of these hormones and proteins is crucial for breast tissue health, and their disruption may significantly influence breast cancer development.

The molecular docking analysis revealed that kaempferol binds within the active site region of CYP1B1, occupying a pocket proximal to residues involved in substrate recognition and catalytic activity. The interaction was stabilized by hydrogen bonds and hydrophobic contacts between the hydroxyl groups of kaempferol and key amino acid residues lining the catalytic cavity, suggesting that kaempferol may interfere with substrate access or orientation within the enzyme. Such binding characteristics indicate a potential modulatory effect on CYP1B1-mediated estrogen metabolism rather than nonspecific surface association.

Similarly, docking to HSD17B2 showed that kaempferol interacts within the enzyme's ligand-binding domain, forming stabilizing hydrogen bonds with residues involved in cofactor or substrate binding. The positioning of kaempferol within this region suggests a potential influence on the enzymatic conversion of active estradiol to its less active form, estrone. The presence of multiple non-covalent interactions supports the formation of a stable ligand–protein complex that may affect HSD17B2 catalytic function. Collectively, these interaction patterns indicate that kaempferol may modulate estrogen-related enzymatic activity through direct binding to functional regions of CYP1B1 and HSD17B2, consistent with its proposed multitarget mechanism in breast cancer.

4. CONCLUSION

This study reveals that kaempferol derived from *Moringa oleifera* has potential anticancer properties against breast cancer, as evidenced by predicted molecular interactions with estrogen-metabolism-related proteins CYP1B1 and HSD17B2. Kaempferol's physicochemical properties indicate desirable drug-likeness, as it satisfies Lipinski's rule of five. Kaempferol is designated as toxicity class 5, indicating a low acute oral toxicity based on LD_{50} calculations. These toxicity data are computational predictions and do not provide actual evidence of clinical safety or adverse effects. Molecular docking results showed that kaempferol has a low binding affinity for CYP1B1 and HSD17B2. Taken together, these data suggest kaempferol's potential as a lead compound for future research. However, experimental validation through in vitro and in vivo studies is required to validate its biological activity and toxicological profile.

CONFLICT OF INTEREST

All authors declared that there was no conflict of interest.

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