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Unveiling the galactagogue mechanism of *leptadenia reticulata* active components: in-silico molecular docking studies on dopamine, oxytocin, and thyroid hormone receptors.

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ABSTRACT

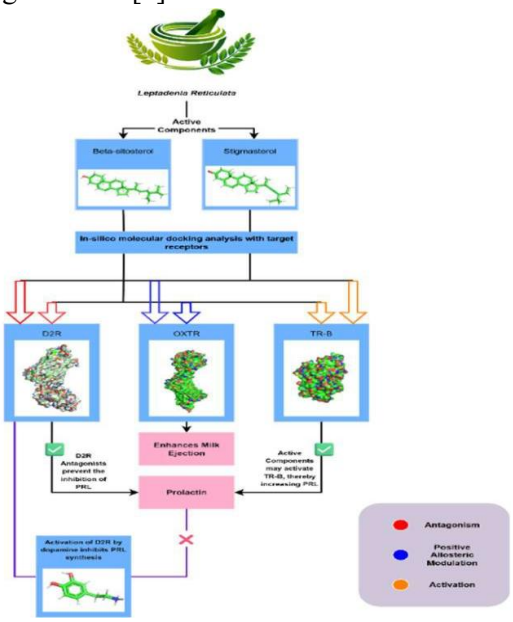
Leptadenia reticulata is traditionally used to enhance lactation; however, its precise molecular mechanism of action remains unclear. To investigate the interactions of two major phytosterols: stigmasterol and β-sitosterol with key lactation-related receptors using in-silico molecular docking. Molecular docking studies were conducted to examine the binding of stigmasterol and β-sitosterol to dopamine D2 receptor (D2R), oxytocin receptor (OXTR), and thyroid hormone receptors (TR_β and TR_α). Both compounds exhibited strong binding affinity to D2R near the dopamine active site, suggesting potential non-competitive inhibition of dopamine's action, which may enhance prolactin secretion. They also bound to OXTR with greater affinity than cholesterol, indicating a possible role in facilitating oxytocin-mediated milk ejection. Moderate interactions with TR_β and TR_α suggest a supportive influence on lactation through thyroid regulation. The study reveals a possible multi-receptor mechanism by which *Leptadenia reticulata* may promote lactation, supporting the application of computational methods to validate traditional therapeutic claims.

Keywords: *Leptadenia reticulata,* stigmasterol, β-sitosterol, Galactagogue, Molecular Docking

1. INTRODUCTION

Leptadenia reticulata is a versatile and traditionally valued medicinal plant, deeply entrenched in Ayurvedic medicine, where it is revered as a *Rasayana* herb known to promote vitality, rejuvenation, and metabolic balance [1]. Over the centuries, it has been widely

employed for its therapeutic benefits across a spectrum of health conditions, and its inclusion in formulations such as Chyawanprash, Speman, Leptaden, Envirocare, and Calshakti attests to its commercial and medicinal significance [2].



Phytochemically, *L. reticulata* possesses a rich composition of bioactive compounds contributing to its broad pharmacological profile. Key constituents include (i) sterols (β-sitosterol, γ-sitosterol, campesterol, stigmasterol), (ii) triterpenoids (α-amyrin, β-amyrin, lupeol, simiarenol), (iii) flavonoids (apigenin, luteolin, diosmetin, rutin, reticulin, deniculatin, leptaculatin), (iv) fatty acid like methyl esters, and (v) other secondary metabolites like ferulic acid, hentriacontanol, leptadenol, leptidin, and β-amyrin acetate. These compounds are associated with diverse bioactivities including antidiabetic, antioxidant, immunomodulatory, anti-inflammatory, anticancer, lactogenic, anti-abortifacient, antimicrobial, and antifungal effects [3,4,5,6]. In addition, vasodilatory, hypotensive, antidepressant, and anti-anaphylactic effects have also been reported, suggesting potential for modern drug development [1,3]. Recent high-resolution LC-MS/MS (Q-TOF) profilin has revealed the presence of 113 phytocompounds in methanolic extracts of L. reticulata, extracted from roots, stems, and leaves. These compounds demonstrated favorable pharmacokinetic properties, including compliance with Lipinski's rule of five, good gastrointestinal absorption, and low toxicity (LD_{50} > 2000 mg/kg), reinforcing their relevance in rational drug design [7]. While traditional uses are well-documented, claims should be verified using an in-silico approach before initiating human clinical trials, especially to establish evidence-based applications for galactagogue (lactation-promoting) effects. Despite the extensive pharmacological claims, the precise molecular mechanisms underlying specific therapeutic actions of L. reticulata, particularly its galactagogue (lactation-promoting) activity, remain largely unexplored. Given that phytosterols such as stigmasterol and β-sitosterol are implicated in hormonal modulation, their potential interactions with lactation-associated receptors warrant investigation. Dopamine D2-receptor (D2R) signaling inhibits lactation by activating D2R on pituitary lactotrophs via Gi, suppressing adenylate cyclase and prolactin gene transcription. D2R agonists (e.g., bromocriptine) reduce prolactin secretion and milk production, while antagonists (e.g., domperidone) promote lactation [8]. D2R on mammary alveolar epithelial cells lowers cAMP, inhibits STAT5 and glucocorticoid signaling, reduces milk protein synthesis (casein, lactoferrin), and induces apoptosis via ERK inactivation [9]. Oxytocin, via OXTR on myoepithelial cells, triggers G_q/Ca^{2+} contraction for milk ejection and accelerates casein vesicle exocytosis, with its absence impairing the milk-letdown reflex in mice, supporting clinical use of nipple stimulation or Pitocin [10]. Thyroid hormone signaling via TR_β enhances lactation by up-regulating $TR_\beta 1$ and type-II deiodinase at parturition, boosting T3 and STAT5 activity, while hypothyroidism reduces prolactin-receptor and α-lactalbumin expression, and hyperthyroidism impairs gland development and milk quality [11,12,13].

Pathway disruptions cause lactation failure. This study aims to elucidate the galactagogue mechanism of *Leptadenia reticulata* by employing in-silico molecular docking approaches to evaluate the binding affinities of its key phytosterols with dopamine, oxytocin, and thyroid hormone receptors. The findings may offer insights into a multi-receptor mediated mechanism supporting the plant's traditional use in enhancing lactation and contribute to the evidence base for its therapeutic applications.

2. MATERIALS AND METHODS

2.1. Target Selection and Pocket Detection

The study targeted three protein receptors relevant to the hypothesized mechanism of action of Leptadenia reticulata galactagogue properties: Dopamine receptor (PDB ID: 6CM4), oxytocin receptor (PDB ID: 6TPK), and Thyroid Hormone Receptors (TRβ) (PDB ID: 3GWS) and (TRα) (PDB ID: 1NAV).

The protein structures were retrieved from the Protein Data Bank (PDB). Binding pocket analysis was performed using the fpocket algorithm to identify potential ligand-binding sites of TRβ and TRα (PDB ID: 3GWS, 1NAV). The first-ranked pocket, based on fpocket scoring criteria (pocket volume and hydrophobicity), was selected for molecular docking studies to ensure focus on the most probable binding site.

2.2. Receptor Preparation

The retrieved PDB structures were pre-processed using AutoDockTools (version 1.5.6). Pre-processing steps included the removal of water molecules, ions, and other non-protein entities to simplify the receptor model. Polar hydrogens were added, and Gasteiger charges were assigned to stabilize the protein's electrostatic environment. The receptor structures were saved in PDB format, compatible with Dockit (https://github.com/aretasg/dockit).

2.3. Ligand Preparation

Two small-molecule compounds from Leptadenia reticulata, stigmasterol (PubChem CID: 5280794) and β-sitosterol (PubChem CID: 222284), were selected as ligands based on their reported galactagogue properties. The 3D conformers of these compounds were retrieved from PubChem. Ligand structures were optimized using Chimera (version 1.18) to minimize energy.

2.4. Molecular Docking

Molecular docking was performed using Dockit (https://github.com/aretasg/dockit) with the AutoDock Vina engine [14]. Prior to docking, all structures were preprocessed by removing water molecules and saving them in PDB format. Both receptor and ligand structures were energy minimized using UCSF Chimera's DockPrep tool. Two types of docking strategies were employed: blind docking and targeted docking. Blind docking was conducted to explore all potential binding sites across the receptor surface. The pose with the lowest binding energy (in kcal/mol) was selected for interaction analysis. In parallel, targeted docking was performed. For targeted docking, pockets were identified using Fpocket [15], and the first-ranked pocket was used for docking in the structures 3GWS and 1NAV. The receptors were initially verified by studying the interaction between native ligands/known inhibitors. For dopamine receptor (D2R) (PDB ID: 6CM4), the interaction complexes of stigmasterol and β-sitosterol with D2R were saved as .pdb files using PyMOL and then dopamine (CID: 681) was docked with these two modified receptors to assess inhibitory action of these compounds. Ligand–receptor interactions such as hydrogen bonds and hydrophobic contacts were visualized using PyMOL (version 2.5.2).

2.5. Software and Computational

Resources

All computational analyses were conducted on a workstation running Ubuntu 20.04 LTS, equipped with an Intel processor (3.6 GHz), 8 GB RAM, and a 4 GB dedicated graphics card. The following software tools were used throughout the study: Fpocket (version 2.0) for pocket detection, Dockit (AutoDock Vina engine) for molecular docking, AutoDockTools (version 1.5.6) for structure preparation, Open Babel (version 3.1.1) for file format conversions, UCSF Chimera for minimization and preprocessing, and PyMOL (version 2.5.2) for visualization and interaction analysis.

3. RESULTS

3.1 Interaction of Stigmasterol and β-Sitosterol with Dopamine receptor D2R

The validation of D2R was first performed by docking Dopamine and D2R. The binding affinity of the complex was ranging from -6.5 kcal/mol to -4.9 kcal/mol for the first 9 poses (Table 1). All the nine poses were observed in PyMOL. The 2D interaction diagram of the first pose (Fig 1) revealed the Interaction of Dopamine with key residues of the active site of D2R such as ASP:80, CYS:84, THR:85, TRP:373 confirming that Dopamine is binding to the active site. Then, the affinities of Stigmasterol and Beta-Sitosterol with D2R were checked. Both stigmasterol and β-sitosterol showed very strong Interaction with D2R (-9.8 kcal/mol and -9.5 kcal/mol) (Table 1). The 2D interaction analysis of both molecules revealed their interaction with some common residues of Dopamine interaction, however, no direct bond formation with the key active site residue ASP:80 is observed (Fig 2, Table 2). This indicates that stigmasterol and β-sitosterol might act as non-competitive inhibitors of the dopamine receptor making the normal interaction between dopamine and D2R difficult.

Table 1

Affinity values (kcal/mol) for top 9 docking poses of D2R-ligand complexes:

Pose	D2r+ Dopa	D2r +stig	D2r +sito	D2r- stig+ Dopa	D2r- sito+ Dopa
1	-6.5	-9.8	-9.5	-5.9	-5.3
2	-6.3	-9.5	-9.4	-5.9	-5.3
3	-5.4	-9.1	-9.1	-5.6	-5.2
4	-5.3	-9.0	-8.6	-5.5	-5.2
5	-5.3	-8.6	-8.3	-5.3	-4.9
6	-5.2	-8.6	-8.3	-5.3	-4.9
7	-5.2	-8.5	-8.3	-5.2	-4.9
8	-5.2	-8.3	-8.2	-5.1	-4.9
9	-5.1	-8.1	-8.2	-5.1	-4.9

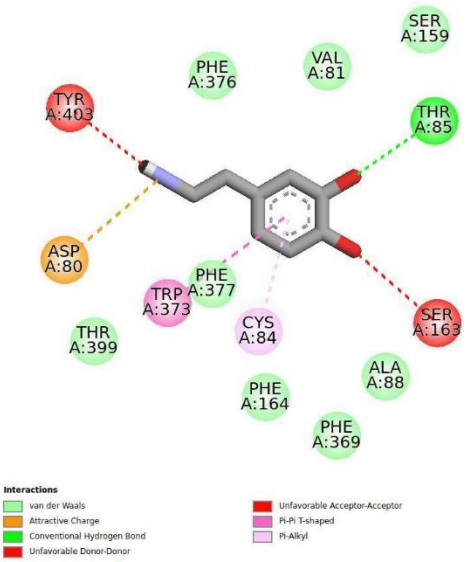


Fig. 1. Interaction of Dopamine with D2R

To confirm their role as non-competitive inhibitors, we incorporated these molecules into the D2R structure using PyMOL and then analyzed the interaction Dopamine with these combined ligands (D2R-Stigma and D2R-Sito). The binding affinities of D2R-Stigma with Dopamine (Table 1) revealed that although there is a decrease in the affinity, Dopamine still is interacting with the active site (Fig 2A, 3A). Whereas upon analysis of D2R-Sito with dopamine, much lesser binding affinities were observed. It was further

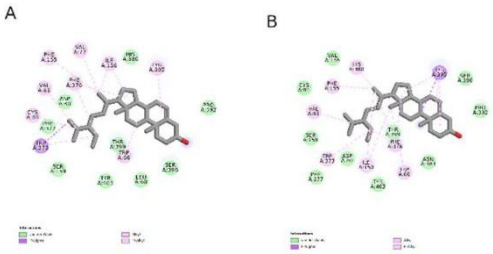


Fig. 2. 2-D Interaction map of stigmasterol with D2R (A) and 2-D Interaction map of β – sitosterol with D2R (B).

Table 2

Analyzed that the ninth pose of Dopamine (-4.9 kcal/mol) was interacting near the active site (Fig 3B). The decrease in binding affinity after incorporation of the ligands suggests that these two ligands may be acting as non-competitive inhibitors of D2R and inhibiting the dopamine interaction, with β-sitosterol being the better inhibitor.

Molecule	Afinity (kcal/mol)	Hydrophobic	H-bond	Unfavourable
Dopamine	-6.5	Trp(A:373); Cys(A:84)	Thr(A:85); Asp(A:80)	Ser(A:163); Tyr(A:403)
Stigmasterol	-9.8	Trp(A:373); Cys(A:84); Ile(A:150); Val(A:81); Phe(A:155); Phe(A:376); His(A:380); Tyr(A:395); Trp(A:66)	None	None
β-sitosterol	-9.5	Trp(A:373); Cys(A:84); Ile(A:150); Val(A:81); Phe(A:155); Phe(A:376); Tyr(A:395); Trp(A:66); Val(A:77)	None	None

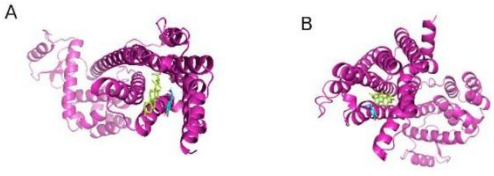


Fig. 3. 3-D Interaction of D2R having stigmasterol (A) and β–sitosterol (B) with Dopamine (blue) (Pose 1 and 9 respectively)

Interaction details for top docking poses of D2R complexes with Dopamine

3.2. Interaction of Stigmasterol and β-Sitosterol with Oxytocin receptor OXTR

We then studied the interaction of stigmasterol and β-sitosterol with Oxytocin receptor OXTR as oxytocin plays an important role in milk ejection. Initially, we validated the receptor by interaction with cholesterol, as cholesterol is known to be a Positive Allosteric Modulator. The binding affinities of the complex are given in Table

2. We then studied the interaction of Stigmasterol and β- Sitosterol with OXTR and found that both of these molecules interact with the OXTR strongly and in the same position as cholesterol (Table 3). The common interacting residues were found to be Pro(A:170); Ile(A:174); and Tyr(A:200).

These findings suggest that stigmasterol and β- sitosterol may act as effective modulators of OXTR, potentially influencing oxytocin-mediated processes

such as milk ejection, with Stigmasterol demonstrating a particularly favorable binding profile compared to the positive allosteric modulator, cholesterol.

Pose	Cholesterol	Stigmasterol	β-sitosterol
1	-8.5	-9.7	-8.2
2	-8.3	-9.6	-8.1
3	-8.3	-9.5	-8.1
4	-8.0	-9.4	-8.1
5	-7.9	-9.4	-8.0
6	-7.8	-9.2	-7.9
7	-7.8	-9.1	-7.9
8	-7.7	-9.0	-7.9
9	-7.7	-8.8	-7.8

Table 3: Affinity values (kcal/mol) for top 9 docking poses of OXTR-ligand complexes

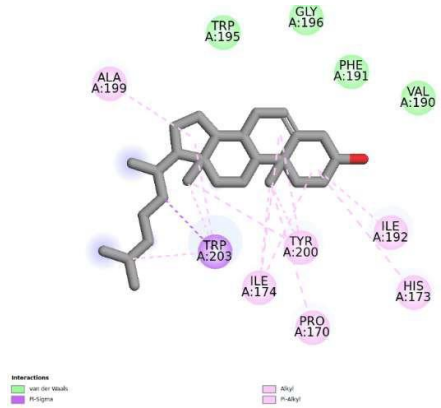


Fig. 4. Interaction of Cholesterol with OXTR.

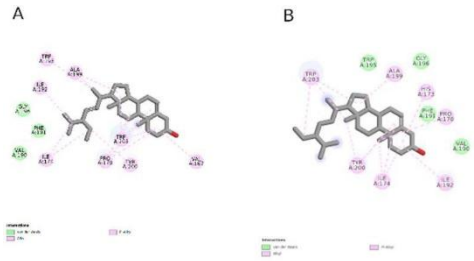


Fig. 5. 2-D Interaction map of stigmasterol (A) and β-sitosterol (B) with OXTR.

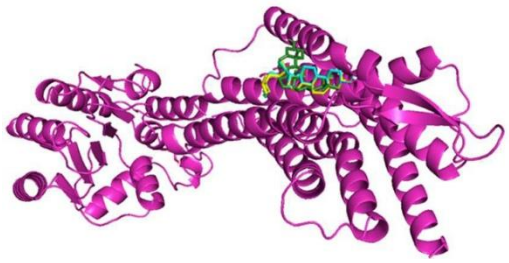


Fig. 6. 3-D Interaction of OXTR with Cholesterol (Blue), Stigmasterol (Green), and β-sitosterol (Yellow).

Table 4: Interaction details for top docking poses of cholesterol, stigmasterol and β-sitosterol with OXTR

Molecule	Affinity (kcal/mol)	Hydrophobic	H-bond	Unfavorable
Cholesterol	-8.5	Trp(A:203); Ile(A:174); Ile(A:192); Pro(A:170); Ile(A:174); Ala(A:199); His(A:173); Try(A:200); Trp(A:203)	None	None
Stigmasterol	-9.7	Trp(A:167); Pro(A:170); Ile(A:192); Ala(A:199); Ile(A:174); Tyr(A:200); Trp(A:203)	None	None
β-sitosterol	-8.2	Ile(A:174); Ala(A:199); Ile(A:192); Pro(A:170); His(A:173); Tyr(A:200); Trp(A:203)	None	None

3.3. Interaction of stigmasterol and β-sitosterol with Thyroid Hormone Receptor (TR_β)

The thyroid hormone plays an important role in the development of mammary glands and thus ultimately, milk formation. We first validated TR_β by interaction with T3. The interactions of T3, stigmasterol and β-sitosterol with TR_α are given in supplementary data (Fig S2, S3, Table S3, S4). The binding affinities are given in table 5 and ranged from -9.2 kcal/mol to -6.8 kcal/mol. The 2-D interaction map revealed the interaction of T3 with some key active site residues such as His435, Arg282, Asn331, Leu330, Met310 (Figure 7, Table 5). A strong interaction with key residues of active site confirmed the receptor-ligand validation. We then studied the interaction of stigmasterol and β-sitosterol with the same active site pocket. The binding affinities of stigmasterol (-7.2 kcal/mol) and β-sitosterol (-7.6 kcal/mol) (Table 5) were moderate as compared to T3.

Table 5: Affinity values (kcal/mol) for top 4 docking modes of TR_β-ligand complexes

Pose	T3	Stigmasterol	β-sitosterol
1	-9.2	-7.2	-7.6
2	-9.2	-7.0	-7.0
3	-7.1	-	-6.7
4	-6.8	-	-

The 2-D interaction map of stigmasterol and sitosterol were analyzed to check common interacting residues (Figure 8A and 8B). The common interacting residues between T3, stigmasterol, and sitosterol are Leu(X:330), Ala(X:279), Arg(X:316), Phe(X:272). Other interacting residues are described in Table (6). We further confirmed whether the interactions of stigmasterol and β-

sitosterol are in the same pocket of T3 by analysing the 3-D interaction map and overlapping all three ligands (Figure 9). This indicates that stigmasterol and β-sitosterol might moderately activate the TR_β receptor and hence have an effect on lactation.

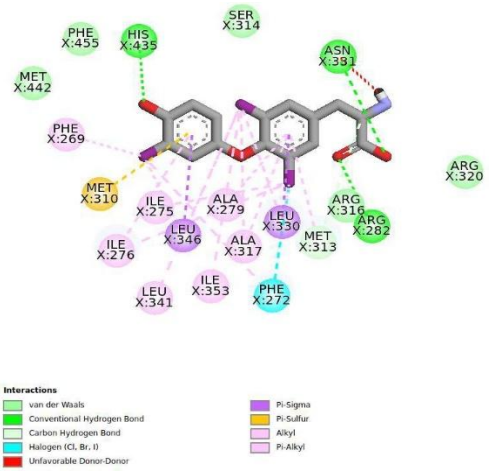


Fig. 7. 2-D interaction map of T3 with TR_β.

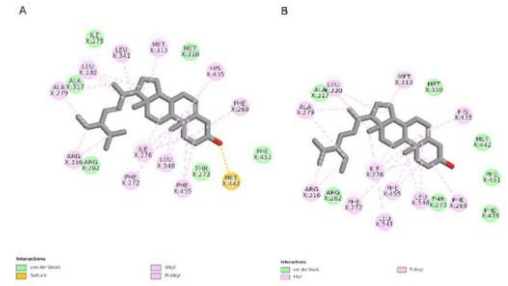


Fig. 8. (A) 2-D Interaction map of stigmasterol (A) and β-sitosterol (B) with TR_β.

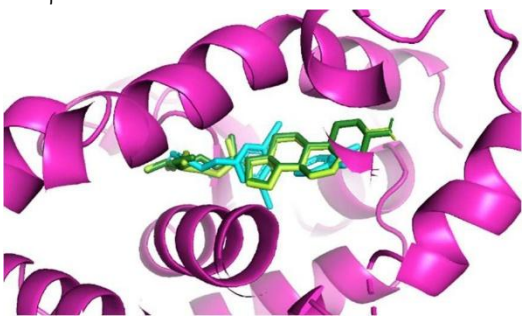


Fig. 9. 3-D interaction of TR_β having T3 (blue), stigmasterol (green) and β-sitosterol (yellow).

4. DISCUSSION

The in-silico molecular docking study of stigmasterol and β-sitosterol with the dopamine D2 receptor (D2R) provides evidence for their role potential non-competitive inhibitors in lactation enhancement. Both compounds demonstrated strong binding affinities (-9.8 kcal/mol for stigmasterol and -9.5 kcal/mol for β-sitosterol) compared to dopamine (-6.5 kcal/mol). Dopamine interacts with key active site residues such as ASP:80, CYS:84, THR:85 and TRP:373 [16],

Table 6: Interaction details for top docking modes of T3, stigmasterol and β-sitosterol with TR_β

Molecule	Afinity (kcal/mol)	Hydrophobic	H-bond	Halogen	Unfavorable
T3	-9.2	Phe(X:269); Ile(X:275); Ile(X:276); Leu(X:346); Leu(X:341); Ala(X:279); Ala(X:317); Ile(X:353); Leu(X:330);	Asn(X:331); Arg(X:282); His(X:435);	Phe(X:272)	Asn(X:331)
stigmasterol	-7.2	Phe(X:268); Leu(X:330); Met(X:313); Ala(X:279); Arg(X:316); Ile(X:276); Phe(X:272); Leu(X:346); Phe(X:455); His(X:435); Leu(X:341)	None	None	None

β-sitosterol	-7.6	His(X:435); Met(X:313); Leu(X:330); Ala(X:279); Arg(X:316); Phe(X:272); Leu(X:341); Ile(X:276); Phe(X:455); Leu(X:346); Phe(X:269)	None	None	None

whereas stigmasterol and β-sitosterol bind near the active site, interacting with residues like TRP:373 and CYS:84 but not ASP:80. This binding pattern reduces dopamine's affinity to D2R (-5.9 kcal/mol with stigmasterol and -5.3 kcal/mol with β-sitosterol), suggesting competitive inhibition. Interaction of dopamine with D2R results in suppression prolactin release, a critical hormone for lactation [17], the non-competitive inhibitory activity of these compounds likely diminishes this inhibition, promoting prolactin secretion and thus enhancing milk production. β-Sitosterol appears to be a more effective inhibitor, as it causes a greater reduction in dopamine's binding affinity.

For the oxytocin receptor (OXTR), stigmasterol and β-sitosterol exhibited strong binding affinities (-9.7 kcal/mol and -8.2 kcal/mol, respectively), comparable to or exceeding that of cholesterol (-8.5 kcal/mol), a known positive allosteric modulator [18]. Both compounds interacted with key residues such as PRO:170, ILE:174, and TYR:200, mirroring cholesterol's binding site, which suggests they may act as positive allosteric modulators of OXTR, like cholesterol. Oxytocin is essential for milk ejection [19], and the favorable binding profiles of stigmasterol and β-sitosterol indicate they could enhance oxytocin-mediated signaling, facilitating milk release during lactation. Stigmasterol's higher binding affinity suggests it may be particularly effective in modulating OXTR activity, potentially amplifying the milk ejection reflex. These findings align with the galactagogue properties of Leptadenia reticulata, supporting its traditional use in promoting lactation.

The interaction of stigmasterol and β-sitosterol with thyroid hormone receptor (TR_β) showed moderate binding affinities -7.2 kcal/mol and -7.6 kcal/mol for TR_β) compared to the native ligand T3 (-9.2 kcal/mol for TR_β). Both compounds interacted with residues near the active site, such as HIS:435, ARG:282 and LEU:330, [20,21]. suggesting that they may weakly influence thyroid hormone signaling. However, this interaction might be more important for people with hypothyroidism. Thyroid hormones regulate metabolism and energy balance, which indirectly support lactation by maintaining maternal health and milk synthesis [22]. The combined effect of these compounds on D2R, OXTR, and TRs likely enhances lactation synergistically: reduced dopamine inhibition via D2R increases prolactin levels, enhanced OXTR activity promotes milk ejection, and TR modulation supports metabolic demands. Together, these mechanisms suggest that *Leptadenia reticulata*, containing stigmasterol and β-sitosterol, could effectively boost milk production and release, validating their traditional use as galactagogues. This in-silico molecular docking approach provides valuable insights on the potential galactagogue mechanisms of stigmasterol and β-sitosterol from Leptadenia reticulata, but it has limitations that warrant consideration. The study relies on computational predictions, which, while robust, require validation through in-vivo and clinical studies to confirm the efficacy and safety of *Leptadenia reticulata* in enhancing lactation. Compared to established pharmaceutical galactagogues like domperidone, which directly antagonizes D2R to increase prolactin levels, stigmasterol and β-sitosterol's non-competitive inhibition may offer a gentler, plant-based alternative with

potentially fewer side effects, though their potency remains untested in clinical settings [17]. Additionally, the moderate binding affinities to thyroid hormone receptors suggest a supportive role in maternal metabolism, but their impact may be more pronounced in hypothyroid patients, necessitating targeted studies. Alarsin, a company currently manufactures Leptaden Tablets, containing a mixture of *Leptadenia reticulata* and *Breynia patens*. These tablets have been used to increase lactation in humans as well as cattle [23,24]. Future research should explore the interactions of *Breynia patens*, dose-response relationships, potential synergistic effects with other *L. reticulata* compounds, and long-term safety profiles to establish clinical applicability, particularly for breastfeeding mothers seeking natural lactation support.

5. CONCLUSION

The present study offers a preliminary but insightful computational framework for understanding the lactogenic potential of *Leptadenia reticulata* derived compounds. By identifying key binding interactions of stigmasterol and β-sitosterol with receptors involved in prolactin regulation, milk ejection, and metabolic support, this work lays the foundation for mechanistic validation of traditional herbal formulations like *Leptadenia reticulata* extracts. These findings highlight not only the therapeutic promise of phytosterols but also the utility of in-silico docking in deciphering polyherbal modes of action within complex biological systems. While molecular docking provides a static view of ligand-receptor interactions, it does not capture the full spectrum of dynamic behaviour within biological environments. Future studies should incorporate molecular dynamics simulations (MDS) to evaluate the stability, flexibility, and conformational changes of these ligand-receptor complexes over time. Such simulations would offer deeper insights into binding kinetics and help prioritize compounds for experimental validation. Coupling MDS with in vitro assays and pharmacokinetic profiling will be essential to move from theoretical predictions to translational potential in lactation support.

Author Contributions

NA and TG contributed in conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft and visualization. ST contributed in validation, investigation, data curation and visualization. UKP contributed in writing, review and editing, supervision and project administration. RC contributed in methodology, resources, writing, review and editing, supervision and project administration.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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