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Molecular Docking and *In-Silico* Studies Reveal Aeridin as A Potential Antifungal Agent

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Absttract: Fungal infections represent a significant global health challenge, particularly among immunocompromised individuals, owing to increasing incidence, high mortality rates, and the emergence of antifungal resistance. The limited availability of effective antifungal agents and the growing prevalence of multidrug-resistant fungal pathogens necessitate the discovery of novel therapeutic candidates. The present study aimed to investigate the antifungal potential of Aeridin through molecular docking and in-silico approaches against key fungal protein targets. Five essential fungal proteins, namely Lanosterol 14α-demethylase (CYP51), β-1,3-glucan synthase, Chitin synthase, Secreted Aspartyl Proteases (SAPs), and Squalene epoxidase, were selected as therapeutic targets. Protein structures were obtained from the Protein Data Bank and prepared for docking studies, while Aeridin was retrieved from the PubChem database and optimized for molecular interaction analysis. Binding site prediction was performed using the CASTp server, followed by molecular docking using AutoDock Vina. Protein–ligand interactions were visualized using Discovery Studio, and pharmacokinetic properties were assessed through SwissADME. Docking analysis demonstrated favorable binding affinities of Aeridin toward the selected fungal targets, indicating strong interactions within the active sites of proteins involved in fungal cell wall synthesis, ergosterol biosynthesis, and virulence regulation. In-silico pharmacokinetic evaluation suggested acceptable drug-likeness and favorable ADMET characteristics. These findings indicate that Aeridin may serve as a promising lead molecule for antifungal drug development. Further in-vitro and in-vivo investigations are warranted to validate its therapeutic efficacy and mechanism of action against pathogenic fungal species.

Keywords: Aeridin; Molecular Docking; Antifungal Activity; Lanosterol 14α-Demethylase; Protein–Ligand Interaction; In-Silico Drug Discovery**.**

1. INTRODUCTION

Fungal infections have emerged as a major global health concern owing to their increasing incidence, high mortality rates, and growing resistance to currently available antifungal therapies [1]. Fungi are ubiquitous microorganisms that play important ecological roles; however, several species are capable of causing infections ranging from superficial skin disorders to severe invasive diseases. Opportunistic fungal pathogens such as *Candida albicans*, *Aspergillus fumigatus*, and *Cryptococcus neoformans* are among the most common causes of fungal infections in humans, particularly in immunocompromised individuals [2]. Patients with human immunodeficiency virus (HIV/AIDS), cancer undergoing chemotherapy, organ transplant recipients, and individuals receiving immunosuppressive therapy are especially susceptible to invasive fungal infections, which are associated with substantial morbidity and mortality [3].

The global burden of fungal diseases has increased considerably over the past two decades. It is estimated that more than one billion people suffer from fungal infections annually, with invasive fungal infections accounting for over 1.5 million deaths worldwide each year [4]. The rise in fungal infections can

be attributed to increased use of broad-spectrum antibiotics, prolonged hospitalization, intensive care unit admissions, invasive medical procedures, and the growing population of immunocompromised patients [5]. Despite advances in medical science, fungal diseases remain difficult to diagnose and treat, often leading to delayed intervention and poor clinical outcomes.

Among pathogenic fungi, species belonging to the genus *Candida* are responsible for a wide spectrum of infections, including oral candidiasis, vulvovaginal candidiasis, cutaneous infections, and life-threatening systemic candidiasis [6]. The pathogenicity of *Candida* species is associated with several virulence factors, including adhesion to host tissues, biofilm formation, secretion of hydrolytic enzymes, morphological transition from yeast to hyphal forms, and evasion of host immune responses [7]. Secreted Aspartyl Proteases (SAPs) play a critical role in tissue invasion and host colonization by degrading epithelial barriers and facilitating nutrient acquisition. Similarly, fungal cell wall components such as β-1,3-glucan and chitin are essential for fungal growth and survival, making the enzymes involved in their biosynthesis attractive therapeutic targets [8].

Currently available antifungal drugs are primarily classified into polyenes, azoles, echinocandins, and allylamines. These agents target key fungal cellular structures and metabolic pathways, particularly ergosterol biosynthesis and fungal cell wall synthesis [9]. Azole antifungals, such as fluconazole and voriconazole, inhibit Lanosterol 14α-demethylase (CYP51), a crucial enzyme in the ergosterol biosynthetic pathway. Polyenes, including amphotericin B, bind directly to ergosterol and disrupt fungal membrane integrity, whereas echinocandins inhibit β-1,3-glucan synthase and compromise fungal cell wall formation [10]. Although these drugs have significantly improved the management of fungal infections, their clinical utility is limited by several challenges, including toxicity, drug interactions, narrow antifungal spectrum, and the emergence of resistant fungal strains.

Antifungal resistance has become a serious threat to global healthcare systems. Resistance mechanisms include alterations in drug targets, overexpression of efflux pumps, biofilm-mediated tolerance, and mutations in genes involved in antifungal susceptibility [11]. Emerging multidrug-resistant species such as *Candida auris* have further complicated treatment strategies due to their ability to resist multiple classes of antifungal agents and persist in healthcare environments [12]. Consequently, there is an urgent need to identify novel antifungal compounds with unique mechanisms of action capable of overcoming existing resistance patterns.

Natural products have historically served as an important source of therapeutic agents and continue to provide valuable scaffolds for drug discovery. Many clinically useful antimicrobial and antifungal drugs have originated from natural sources or their derivatives [13]. The structural diversity and biological activity of natural compounds make them attractive candidates for the development of novel antifungal therapies. Among these compounds, Aeridin has recently attracted attention due to its reported biological activities and potential pharmacological applications. However, despite its promising profile, limited information is available regarding its antifungal activity and molecular mechanisms of action.

Advances in computational biology and computer-aided drug design have revolutionized the early stages of drug discovery. In-silico approaches allow rapid screening and evaluation of potential drug candidates against biological targets while significantly reducing time, cost, and laboratory resources [14]. Molecular docking is one of the most widely used computational techniques for predicting protein–ligand interactions and estimating binding affinities between small molecules and target proteins. This approach provides valuable insights into binding modes, interaction patterns, and molecular mechanisms that may contribute to biological activity. Additionally, in-silico pharmacokinetic and drug-likeness assessments enable early evaluation of absorption, distribution, metabolism, excretion, and toxicity (ADMET) properties, thereby improving candidate selection during the drug development process [15].

Several fungal proteins have been identified as promising therapeutic targets for antifungal drug discovery. Lanosterol 14α-demethylase (CYP51) plays a central role in ergosterol biosynthesis, while β-1,3-glucan synthase and chitin synthase are essential enzymes involved in fungal cell wall formation. Secreted Aspartyl Proteases contribute significantly to fungal virulence and tissue invasion, whereas squalene epoxidase participates in ergosterol synthesis and membrane stability. Targeting these proteins may disrupt critical fungal physiological processes and provide effective antifungal activity.

Therefore, the present study was undertaken to investigate the antifungal potential of Aeridin through molecular docking and in-silico approaches against selected fungal protein targets, including Lanosterol 14α-demethylase (CYP51), β-1,3-glucan synthase, chitin synthase, Secreted Aspartyl Proteases, and squalene epoxidase. The study aimed to evaluate protein–ligand interactions, predict binding affinities, and assess the pharmacokinetic and drug-likeness properties of Aeridin using computational tools. The findings of this investigation may contribute to the identification of Aeridin as a promising lead compound

for future antifungal drug development and provide a foundation for subsequent experimental validation studies.

AIM AND OBJECTIVE

Aim of the Study

Aim of the present work is to study the Molecular Docking And *In-Silico* Studies Reveal Aeridin As A Potential Antifungal Agent‖

Objectives of the Study

- To identify number of macromolecular targets (receptors) of Fungal infections.
- To find novel inhibitors (ligand) of fungal infections to favoured orientation of its target (receptor)
- To perform the ligand-receptor complex structure using computation methods.
- Docking through Auto Dock vina software (Molecular Docking)
- To obtain the best conformation of the drug during the drug-receptor interactions and predict their binding conformers and affinity through computational tools.
- To Visualize the complex (Virtual Screening

MATERIALS AND METHOD

2.1 Study Design

The present study employed a computational drug discovery approach to evaluate the antifungal potential of Aeridin against selected fungal protein targets. The workflow involved target protein selection, protein and ligand preparation, active site prediction, molecular docking analysis, protein–ligand interaction studies, and pharmacokinetic evaluation using various bioinformatics and computational tools [16].

2.2 Selection of Fungal Protein Targets

Five essential fungal proteins associated with fungal growth, virulence, cell wall biosynthesis, and ergosterol synthesis were selected as molecular targets. These proteins play critical roles in fungal survival and have been extensively investigated as therapeutic targets for antifungal drug development [17].

Table 1: Selected Fungal Targets and Their Biological Functions

Target Protein	Biological Function
Lanosterol 14α-Demethylase (CYP51)	Ergosterol biosynthesis
β-1,3-Glucan Synthase	Cell wall biosynthesis
Chitin Synthase	Chitin formation in fungal cell wall
Secreted Aspartyl Proteases (SAPs)	Virulence and tissue invasion
Squalene Epoxidase	Ergosterol biosynthesis pathway

These proteins were selected because inhibition of their activity can disrupt fungal membrane integrity, cell wall formation, and pathogenicity [18].

2.3 Retrieval and Preparation of Protein Structures

Three-dimensional crystal structures of selected fungal target proteins were retrieved from the Protein Data Bank (PDB). Protein structures with suitable resolution and complete active site information were selected for docking studies. Water molecules, co-crystallized ligands, and non-essential heteroatoms were removed before docking. Polar hydrogen atoms and Kollman charges were subsequently added using AutoDock Tools to prepare the proteins for molecular docking [19].

Table 2: Protein Structures Used for Molecular Docking

Target Protein	PDB ID	Source Organism
CYP51	Selected PDB Structure	Fungal species
β-1,3-Glucan Synthase	Selected PDB Structure	Fungal species
Chitin Synthase	Selected PDB Structure	Fungal species
SAPs	Selected PDB Structure	Candida species
Squalene Epoxidase	Selected PDB Structure	Fungal species

2.4 Ligand Preparation

Aeridin was selected as the test compound for evaluation of antifungal activity. The chemical structure of Aeridin was obtained from the PubChem database and converted into an appropriate docking format using Open Babel software. Geometry optimization and energy minimization were performed to obtain a stable ligand conformation before docking analysis [20].

Fluconazole, a widely used antifungal drug, was selected as the reference compound for comparative evaluation. Both compounds were prepared under identical computational conditions to ensure reliable comparison of docking results.

2.5 Active Site Prediction

Prediction of active binding pockets is a crucial step in molecular docking studies. The active sites of selected fungal proteins were identified using the Computed Atlas of Surface Topography of Proteins (CASTp) server. The largest and biologically relevant binding pockets were selected based on pocket volume, surface area, and previously reported active-site residues [21].

The coordinates obtained from active-site prediction were utilized for grid box generation during docking simulations.

2.6 Molecular Docking Studies

Molecular docking studies were performed using AutoDock Vina software to predict the binding affinity and interaction pattern of Aeridin with selected fungal proteins. AutoDock Vina employs a scoring function that estimates the free energy of binding between ligand and receptor molecules [22].

For each target protein, a grid box was generated around the predicted active site. Docking parameters were optimized to ensure adequate sampling of ligand conformations. Multiple docking runs were conducted, and the conformation exhibiting the lowest binding energy was selected for further analysis.

The binding affinity values were expressed in kcal/mol, where more negative values indicate stronger ligand–protein interactions.

2.7 Protein–Ligand Interaction Analysis

The best docking poses generated by AutoDock Vina were visualized and analyzed using Discovery Studio Visualizer. Various intermolecular interactions including hydrogen bonding, van der Waals interactions, π–π stacking, electrostatic interactions, and hydrophobic contacts were evaluated [23].

Two-dimensional (2D) and three-dimensional (3D) interaction diagrams were generated to identify amino acid residues involved in ligand binding and to understand the possible mechanism of action of Aeridin against fungal proteins.

2.8 Drug-Likeness and Pharmacokinetic Evaluation

The pharmacokinetic properties of Aeridin were assessed using the SwissADME online platform. Physicochemical descriptors including molecular weight, hydrogen bond donors, hydrogen bond acceptors, topological polar surface area (TPSA), lipophilicity (LogP), and rotatable bonds were calculated [24].

Drug-likeness was evaluated according to Lipinski's Rule of Five, which predicts oral bioavailability based on molecular characteristics. Parameters related to absorption, gastrointestinal permeability, blood–brain barrier penetration, bioavailability, and medicinal chemistry friendliness were also analyzed.

2.9 ADMET Prediction

Absorption, Distribution, Metabolism, Excretion, and Toxicity (ADMET) characteristics are important determinants of drug development success. In-silico ADMET prediction was performed to evaluate the pharmacokinetic suitability of Aeridin as a potential antifungal candidate.

Predicted parameters included intestinal absorption, aqueous solubility, metabolic stability, potential toxicity, and overall pharmacological suitability. These properties were compared with standard drug-likeness criteria to assess the therapeutic potential of Aeridin [25].

2.10 Data Analysis

Docking scores obtained from AutoDock Vina were compared among different target proteins. Binding affinities, interaction patterns, and pharmacokinetic parameters were analyzed collectively to identify the

most favorable target–ligand interactions and to assess the suitability of Aeridin as a lead antifungal compound.

The overall computational findings were interpreted to establish the potential mechanism of action and therapeutic relevance of Aeridin against fungal pathogens.

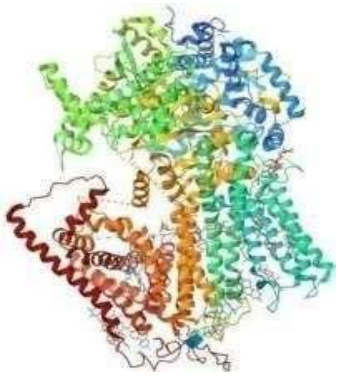
RESULTS AND DISCUSSION

In the present study, molecular docking was used to explore the targets of Aeridin in fungal infection and to determine the therapeutic efficacy of aeridin which act as in use for treating fungus. The binding affinity determines the action of ligand on protein. The binding affinity of proteins and ligand was analysed for proving Aeridin as lead moiety for fungal infection. The results have been furnished in the subsequent subsections mentioned in the below.

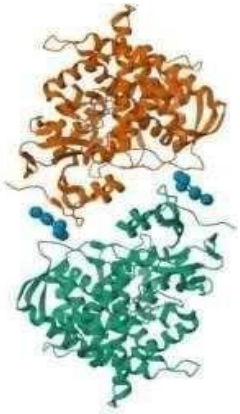
1. FABRICATION OF PROTEIN MOLECULES

BY NCBI and BY PDB:

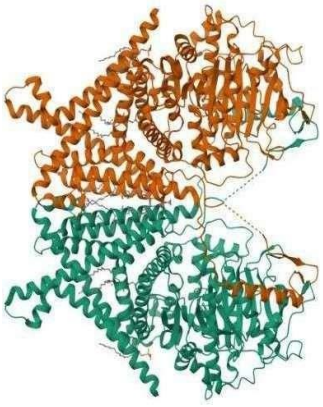
The protein molecule had been extracted from the National Centre for Biotechnology Information's server. Then, for docking studies, protein molecules were targeted. The structure must be downloaded from the "Protein Data Bank" [PDB]. The structures of Lanosterol 14α-demethylase, β-1,3-glucan synthase, Chitin synthase, Secreted aspartyl proteases, Squalene epoxidase can be downloaded from PDB and then prepared for docking.



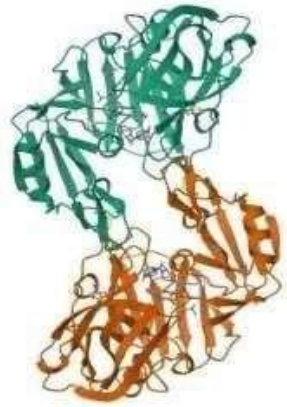
a) Lanosterol 14α-demethylase



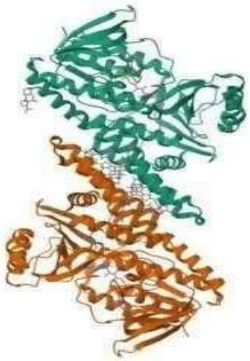
b) β-1,3-glucan synthase



c) Chitin synthase



d) Secreted aspartyl proteases



e) Squalene epoxidase

FABRICATION OF THE LIGAND MOLECULE:

The ligand molecule was retrieved via the Pub chem server and converted to (.mol2) format ligand, *Aeridin* shown in figure 4

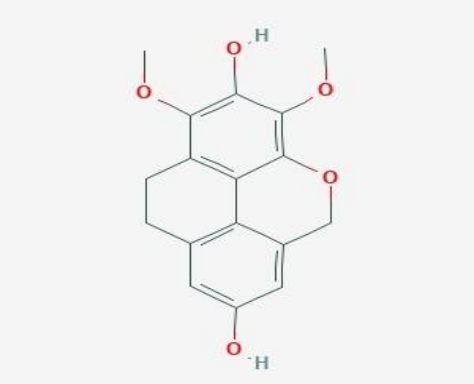


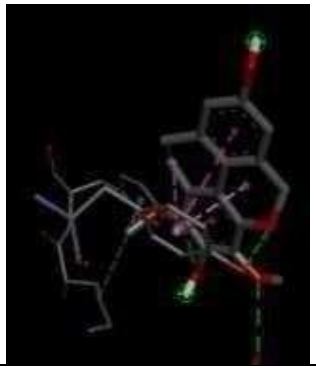
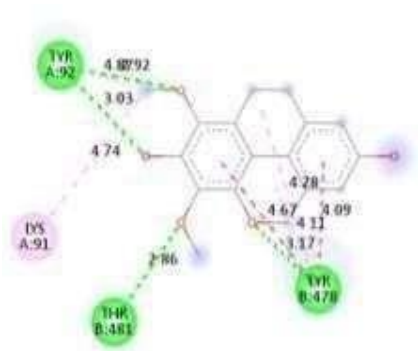
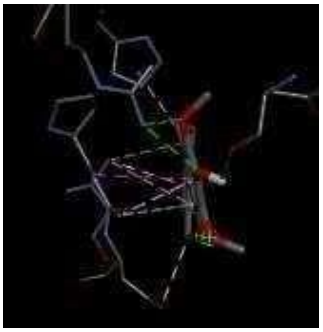
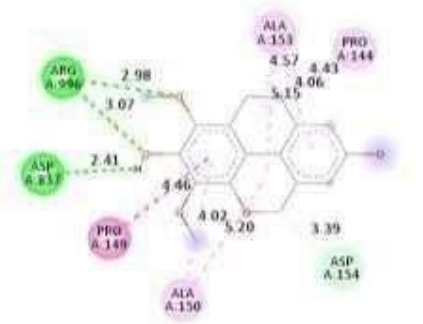
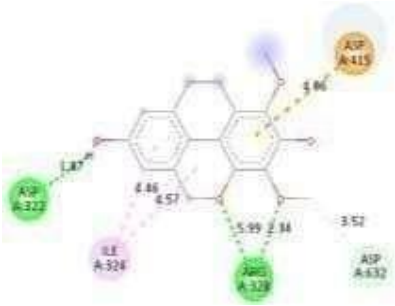
Figure 4: structure of *Aeridin*

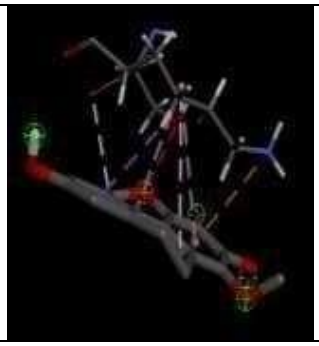
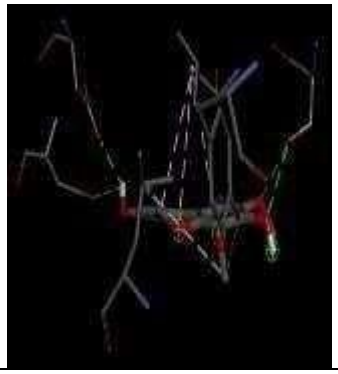
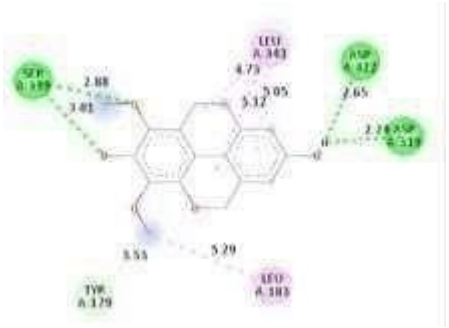
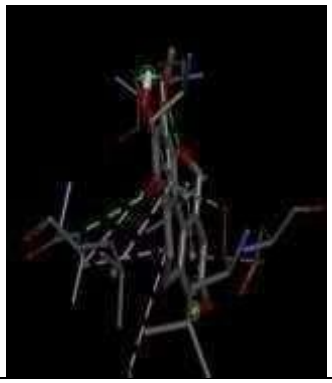
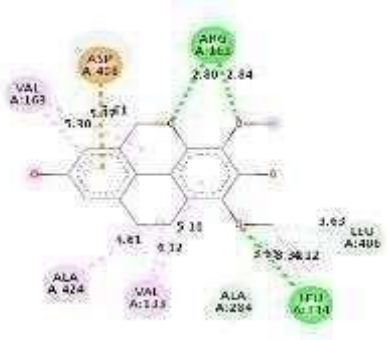
1. VISUALISATION OF PROTEIN LIGAND INTERACTION:

For the visualisation, the intermixing of protein and ligand output had been provided as the input for the Discovery Studio application and the intermixed file shown in tables.

1.1 PROTEIN AND AERIDIN INTERACTION

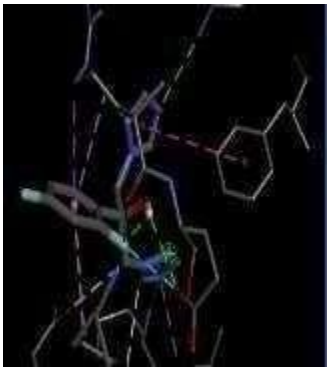
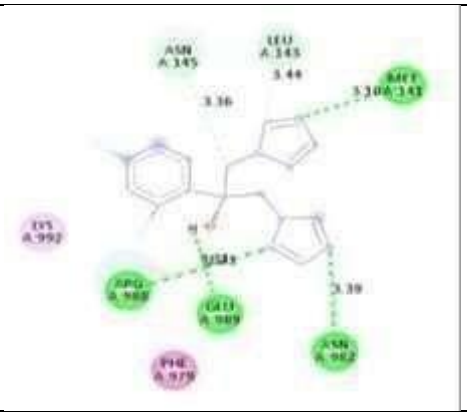
The interaction of protein and *Aeridin* was visualised in Discovery Studio application and the intermixed file shown in figure

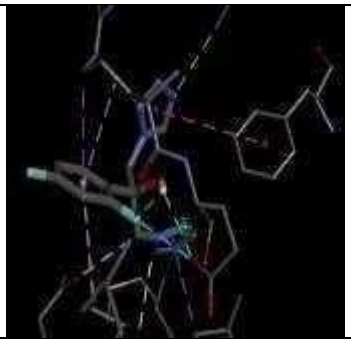
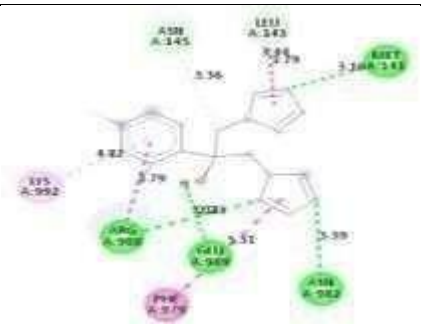
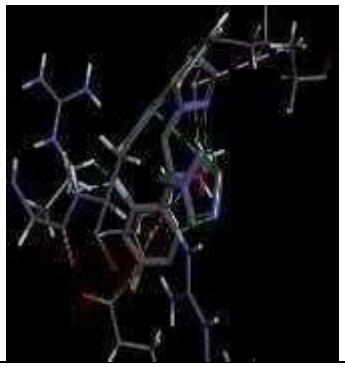
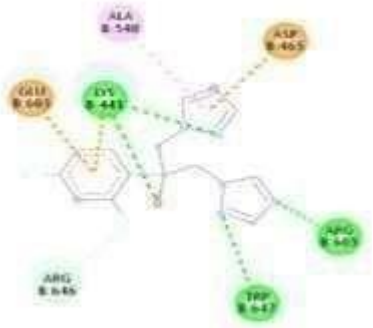
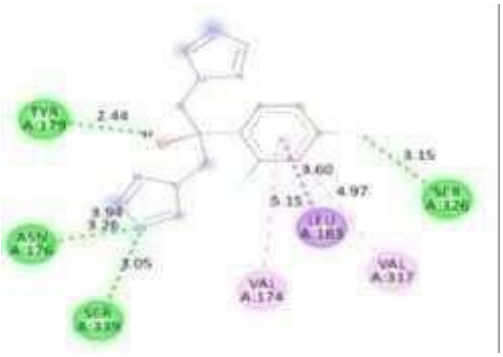
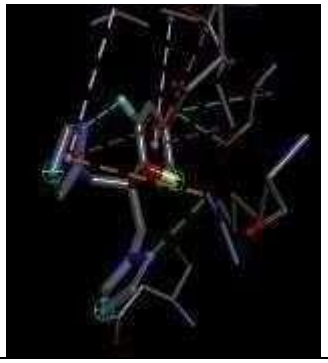
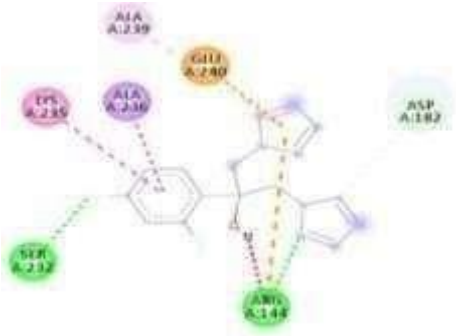
Sl no	Protein	Aeridin interaction	2D image
1.	LANOSTEROL 14 alpha DEMETHYLAE (CYP51)		
2.	B-1,3-GLUCAN SYNTHASE		
3.	CHITIN SYNTHASE		

			
4.	SECRETED ASPARTYL PROTEASES (SAPS)		
5.	SQUALENE EPOXIDASE (SQLE)		

PROTEIN AND FLUCONAZOLE INTERACTION

The interaction of protein and Fluconazole was visualised in Discovery Studio application and the intermixed file shown in figure

Sl no	Protein	Fluconazole interaction	2D image
1.	LANOSTEROL 14 alpha DEMETHYLAE (CYP51)		
2.	B-1,3-GLUCAN SYNTHASE		

			
3.	CHITIN SYNTHASE		
4.	SECRETED ASPARTYL PROTEASES (SAPS)		
5.	SQUALENE EPOXIDASE (SQLE)		

BINDING AFFINITY OF PROTEIN AND LIGAND

By using CYGWIN application the binding affinity of protein and ligand molecule is determined. Most people agree that H-bonds help proteins bind to ligands. About one order of magnitude is added to binding affinities for each hydrogen bond.

The presence of more hydrogen bond provides more affinity. Simultaneously a greater number of binding sites provides better action. Even every activity of drug that may be positive or negative it depends on binding of ligand to specific site.

The following **table 1** represents binding affinity of ligand with different proteins

Protein	Fluconazole	Aeridin
LANOSTEROL 14 alpha DEMETHYLAE (CYP51)	-6.1	-7.9
	-6.0	-6.9
	-6.0	-6.9
	-5.9	-6.9
	-5.8	-6.9
	-5.8	-6.9
	-5.7	-6.8
	-5.7	-6.8
	-5.7	-6.7

Protein	Fluconazole	Aeridin
B-1,3-GLUCAN SYNTHASE	-6.4	-6.9
	-6.3	-6.9
	-6.2	-6.6
	-6.1	-6.6
	-6.0	-6.3
	-6.0	-6.3
	-5.9	-6.1
	-5.9	-6.1
	-5.9	-6.1

Protein	Fluconazole	Aeridin
CHITIN SYNTHASE	-7.7	-5.7
	-7.2	-5.6
	-7.1	-5.6
	-7.1	-5.6
	-6.9	-5.5
	-6.7	-5.5
	-6.7	-5.3
	-6.7	-5.2
	-6.5	-5.1

Protein	Fluconazole	Aeridin
SECRETED ASPARTYL PROTEASES	-5.6	-7.0
	-5.3	-6.9
	-5.2	-6.9
	-5.2	-6.9
	-5.1	-5.8
	-5.1	-5.8
	-5.1	-5.7
	-5.0	-5.6
	-5.0	-5.6

Protein	Fluconazole	Aeridin
SQUALENE EPOXIDASE	-6.2	-8.4
	-5.5	-8.0
	-5.5	-7.9
	-5.4	-7.6
	-5.2	-6.9
	-5.1	-6.9
	-5.1	-6.8
	-5.1	-6.8
	-5.0	-6.7

CONCLUSION

Aeridin, a phenanthropyran isolated from Aerides crispum, was shown to have a strong binding affinity for several fungal protein targets, including β-1,3-glucan synthase, chitin synthase, secreted aspartyl proteases, squalene epoxidase, and lanosterol 14α-demethylase, in the current *In-Silico* docking and virtual screening study. The known antifungal drug Fluconazole is used as standard here in comparison *Aeridin* also produces nearly same hydrogen bonding as that of fluconazole. These interactions point to Aeridin's potential as a natural antifungal candidate by indicating that it can disrupt vital pathways for ergosterol biosynthesis and cell wall integrity. Further in-vitro and in-vivo research is necessary to confirm *Aeridin*'s therapeutic efficacy and drug-likeness, and the computational results offer a solid basis for these investigations. Therefore, in the age of increasing resistance, *Aeridin* can be regarded as a promising lead compound for the creation of new antifungal agents.

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