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In silico antifungal effect of the bioactive compound of *Eucalyptus camadalensus* using molecular docking approach

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ABSTRACT

Stigmasterol, a naturally occurring *phytosterol*, from *Eucalyptus camadalensus* has demonstrated potential pharmacological relevance as a modulator of the *CYP51B* enzyme, a critical component of the sterol biosynthesis pathway in pathogenic fungi. *CYP51B*, a *lanosterol 14α-demethylase*, plays a pivotal role in converting l*anosterol* into *ergosterol*, a key structural element of fungal cell membranes essential for their integrity and function. Targeting *CYP51B* with inhibitors has been an effective antifungal strategy, as seen with azole drugs like *saperconazole* and *pramiconazole*. Computational binding analysis suggests that Stigmasterol exhibits a competitive binding affinity for *CYP51B*, with binding energy values comparable to those of conventional azole antifungals. Molecular interaction analysis reveals that *Stigmasterol* forms a strong hydrogen bond with *Serine* 375, contributing to ligand stabilization within the *CYP51B* active site. Additional stabilization is provided by a carbon-hydrogen bond with *Histidine* 374, while hydrophobic interactions with residues such as *Alanine* 307, Isoleucine 373, and *Leucine* 503 enhance its affinity for the enzyme's hydrophobic binding pocket. Furthermore, π-alkyl interactions with aromatic residues, including *Tyrosine 122, Tyrosine 136, and Phenylalanine* 504, reinforce ligand binding through favorable entropic contributions. This finding implies that *Stigmasterol* may serve as a promising lead compound for antifungal drug development, particularly in response to the increasing prevalence of resistance to synthetic antifungal agents.

Keywords: Molecular Docking; *Eucalyptus camadalensus*; Antifungal; *Stigmasterol*; *Saperconazole*; *Pramiconazole*; CYP51B; Toxicity; LD50.

1. Introduction

Molecular docking has emerged as a powerful computational approach to predict interactions between bioactive compounds and target proteins, providing valuable insights into drug discovery and antifungal research (Morris et al., 2009). One of the key targets in antifungal studies is *CYP51B*, a cytochrome P450 *sterol 14α-demethylase*, which plays a crucial role in ergosterol biosynthesis—a vital component of fungal cell membranes (Becher & Wirsel, 2012). Inhibition of *CYP51B* disrupts fungal cell viability, making it a promising target for developing novel antifungal agents (Watkins et al., 2010).

Sorghum (*Sorghum bicolor*) is a staple cereal crop in Niger Republic, playing a critical role in food security and nutrition (Haussmann et al., 2012). However, sorghum is highly susceptible to contamination by toxigenic fungi, particularly *Aspergillus, Fusarium,* and *Penicillium* species, which produce harmful mycotoxins such as *aflatoxins* and *fumonisins* (Pitt et al., 2013). These mycotoxins pose serious health risks to humans and livestock, leading to economic losses in agricultural production (Bandyopadhyay et al., 2016).

Natural antifungal agents from medicinal plants have gained attention as eco-friendly alternatives to synthetic fungicides. *Eucalyptus camaldulensis*, a widely distributed plant in Africa, is known for its bioactive compounds, including essential oils, *flavonoids*, and *phenolic* compounds, which exhibit antimicrobial and antifungal properties (Djouahri et al., 2013). Recent studies suggest that plant-derived compounds can effectively target fungal *CYP51B*, inhibiting *ergosterol* biosynthesis and reducing fungal growth (Forli et al., 2016).

In this study, we employ molecular docking techniques to investigate the antifungal activity of *Eucalyptus camaldulensis* leaf extract compounds against *CYP51B* in toxigenic fungi affecting sorghum in Niger Republic.

1.1. Study Objectives

The aim of this study is to use the molecular docking techniques to investigate the antifungal activity of *Eucalyptus camaldulensis* leaf extract compounds against *CYP51B* in toxigenic fungi affecting sorghum in Niger Republic:

1) To identify bioactive compounds present in *Eucalyptus camaldulensis* leaf extract with potential antifungal properties.

2) To investigate the molecular interaction between the identified compounds and the CYP51B enzyme, a known antifungal drug target in toxigenic fungi.

3) To evaluate the binding affinity and docking scores of the selected compounds using molecular docking techniques.

4) To determine the potential of the compounds to inhibit CYP51B activity, thereby interfering with fungal growth and development.

5) To explore the antifungal efficacy of these compounds specifically against toxigenic fungi affecting sorghum crops in Niger Republic

2. Methodology

2.1. Molecular Interaction study

The 3D conformers of 33 compounds, derived from the molecular characterisation of plant extracts, and standard inhibitors (*saperconazole and pramiconazole*) were retrieved from PubChem (www.pubchem.ncbi.nlm.nih.gov) (Kim et al., 2016). The crystal structure of the target protein, sterol *14-alpha demethylase* (*CYP51B*) from *Aspergillus fumigatus*, a pathogenic filamentous fungus, was obtained from the Protein Data Bank (PDB) (www.rcsb.org) (Berman et al., 2000) under the accession code 5FRB.

Preliminary preparation of the *CYP51B* protein structure was carried out using Chimera 1.16 (Pettersen et al., 2004), where water molecules and other heteroatoms were removed, followed by docking preparation. The energy of the retrieved compounds was minimised using PyRx 0.8 (Dallakyan & Olson, 2015) to ensure optimal ligand conformations. The docking process was then performed between the ligands and the protein complex.

Post-docking analysis was conducted using Discovery Studio 2021 (BIOVIA, 2020) to visualise and elucidate the molecular interactions between the ligands and *CYP51B*. This included detailed observation of hydrogen bonds, hydrophobic interactions, and other key contacts between the protein and the lead compounds.

2.2. Toxicity study

ProTox 3.0 was used to predict the toxicity and LD50 of the compound the user needs to specify either the name (PubChem name) or canonical SMILES (Simplified Molecular-Input Line Entry System) string of the input compound to run a prediction on the server. Furthermore, the user can also use the drawing box provided by the

(https://www.chemdoodle.com/), to draw user -desired molecular structure. This supports the prediction of even a hypothetical compound before synthesizing it in the laboratory with an accuracy of 70.97% (Banerjee et al., 2024).

3. Results

3.1. Molecular Interactions between top hit compound and *CYP51B*

The molecular docking results between the *CYP51B* protein and top hit compounds as well as standard drugs (Table 1) (*Pramiconazole and Saperconazole)*. *Pramiconazole* (-12kcal/mol) showed a lower binding compared to *Saperconazole* (-11.9kcal/mol) whereas *stigmastero*l (-10.8) showed a slightly higher binding affinity.

Table 1. Binding Affinity of *CYP51B* and top hit bioactive compound

Ligand	Binding Affinity	rmsd/ub	rmsd/lb
Pramiconazole	-12	0	0
Saperconazole	-11.9	0	0
Stigmasterol	-10.8	0	0

Figure 1 shows the molecular interactions between *CYP51B* and *stigmasterol* showing weak van der Waals interaction from *Valine* 135, *Isoleucine* 464, *Phenyalanine* 130 and *Phenyalanine* 234. There exist hydrogen bonds between the oxygen of *stigmasterol* and each of *Serine* 375 and *Histidine* 374. There are nine (9) hydrophobic bonds contributing to the stability of ligand at the active site.

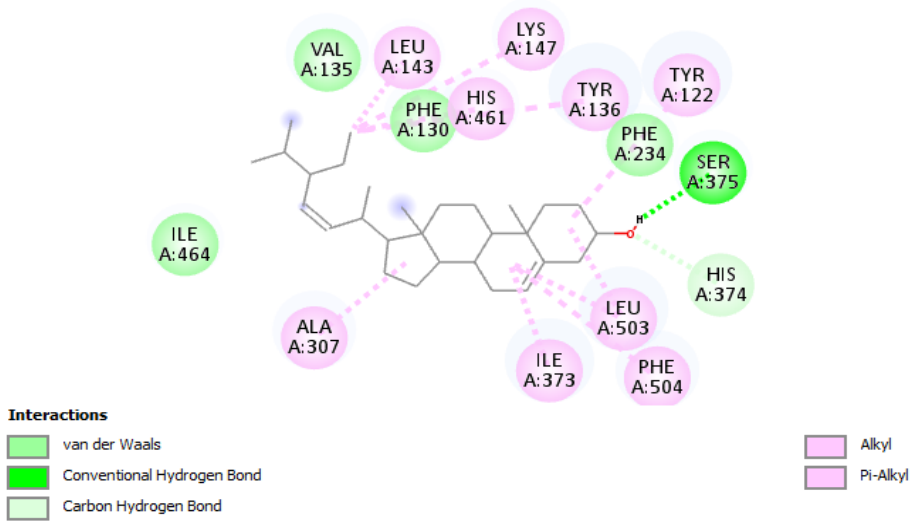

Figure 1. Molecular interaction between *stigmasterol and CYP51B*

An overview of the molecular interactions between *CYP51B* and *Stigmasterol* (Table 2), emphasizing key amino acid residues involved in binding. The interactions include both hydrogen bonding and hydrophobic contacts. Notably, *Stigmasterol* forms a strong conventional hydrogen bond with *Serine 375* (2.27 Å) and a carbon-hydrogen bond with *Histidine 374* (2.76 Å). Additionally, multiple hydrophobic alkyl interactions are observed, including with *Alanine 307* (5.20 Å), *Isoleucine 373* (4.62 Å), and *Leucine 503* (4.93 Å). Other significant hydrophobic interactions include Pi-alkyl interactions involving residues such as *Tyrosine 122* (5.47 Å), *Tyrosine 136* (5.36 Å), *Histidine 461* (4.55 Å), and *Phenylalanine 504* (5.11 Å). These interactions, both hydrogen bonds and hydrophobic

contacts, collectively contribute to the strong binding affinity of *Stigmasterol* to *CYP51B*, suggesting its potential efficacy in modulating the activity of this target protein.

Table 2. Type and length of bonds between *CYP51B* and *Stigmasterol*

From	To	Distance	Categories	Types
Stigmasterol:H	*A: SER375: O*	2.27	Hydrogen	Conventional
A:HIS374:H	*Stigmasterol:O*	2.76	Hydrogen	Carbon Hydrogen
A:ALA307	*Stigmasterol*	5.20	Hydrophobic	Alkyl
A:ILE373	*Stigmasterol*	4.62	Hydrophobic	Alkyl
A:LEU503	*Stigmasterol*	4.93	Hydrophobic	Alkyl
Stigmasterol	*A:LEU503*	5.31	Hydrophobic	Alkyl
Stigmasterol:C	*A:LEU143*	4.48	Hydrophobic	Alkyl
Stigmasterol:C	*A:LYS147*	5.06	Hydrophobic	Alkyl
A:TYR122	*Stigmasterol*	5.47	Hydrophobic	Pi-Alkyl
A:TYR136	*Stigmasterol:C*	5.36	Hydrophobic	Pi-Alkyl
A:HIS461	*Stigmasterol:C*	4.55	Hydrophobic	Pi-Alkyl
A:PHE504	*Stigmasterol*	5.11	Hydrophobic	Pi-Alkyl

- ***Pramiconazole***

The molecular interactions between CYP51B and pramiconazole (Figure 2) which comprises of abundance of weak van der Waals interaction from *Leucine 92, Leucine 91, Threonine 65, Proline 231, Tyrosine 122, Glycine 69, Tyrosine 68, Serine 375, Histidine 374, Isoleucine 377, Phenyalanine 504, Methionine 300, Alanine 307, Histidine 461, Cysteine 463 and Tyrosine 136.* Other interactions include a hydrogen bond with *Leucine* 503.

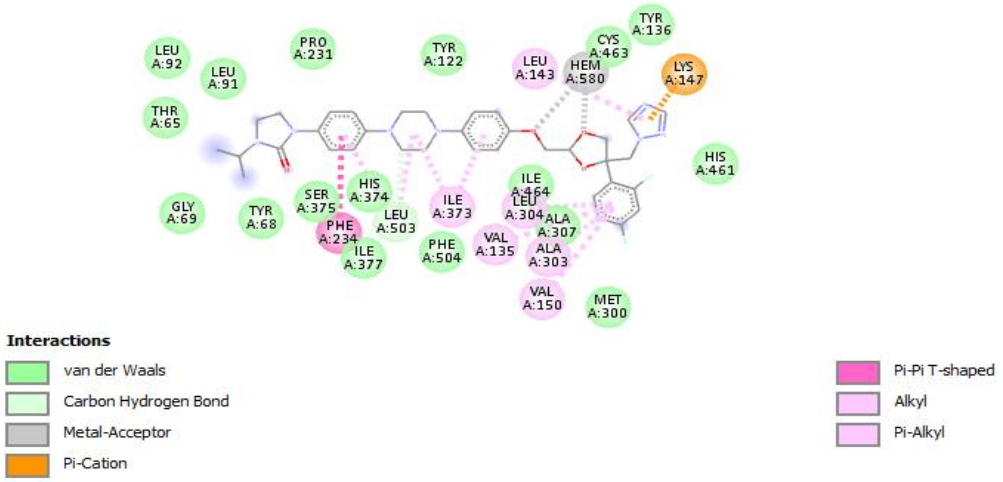

Figure 2. Molecular interactions between *Pramiconazole* and *CYP51B*

The ligand *Pramiconazole* forms a carbon-hydrogen bond with *Leucine 503* (3.52 Å) and a hydrogen bond with *Lysine 147* (2.82 Å), indicating significant stabilizing interactions. Hydrophobic contacts further contribute to the binding, with Pi-Pi T-shaped interactions involving *Phenylalanine 234* (5.04 Å) and several alkyl and Pi-alkyl

interactions with residues such as *Isoleucine 373* (4.71 Å), *Leucine 503* (5.24 Å), *Leucine 304* (4.98 Å), and *Valine 150* (5.31 Å). These diverse interactions, both hydrogen bonds and hydrophobic, suggest that *Pramiconazole* is tightly bound within the active site of *CYP51B*, supporting its potential as an effective ligand for this protein target

The molecular interactions between the target protein *CYP51B* and *saperconazole* (Figure 3). *tyrosine (68, 138), leucine 304, isoleucine (377, 125), phenylalanine (130, 229, 504), threonine (65, 126), and methionine 235*. There are various hydrophobic bonds which include pi-pi and pi-alkyl bonds as well as hydrogen bond between Serine. *Saperconazole* forms a carbon-hydrogen bond with *Serine 375* (3.75 Å) and a significant halogen interaction between its fluorine atom and its own nitrogen (3.57 Å). Hydrophobic interactions are prominent, including Pi-Pi stacked and Pi-Pi T-shaped interactions with *Tyrosine 122* (5.86 Å), *Phenylalanine 234* (5.02 Å), and *Histidine 374* (5.61 Å). Several alkyl and Pi-alkyl interactions are also observed, involving residues like *Isoleucine 373* (4.02 Å and 5.23 Å), *Leucine 91* (3.92 Å), *Leucine 503* (4.85 Å), and *Proline 231* (5.38 Å). These interactions collectively stabilize *Saperconazole* within the protein's active site, emphasizing its potential to effectively bind and inhibit *CYP51B* activity.

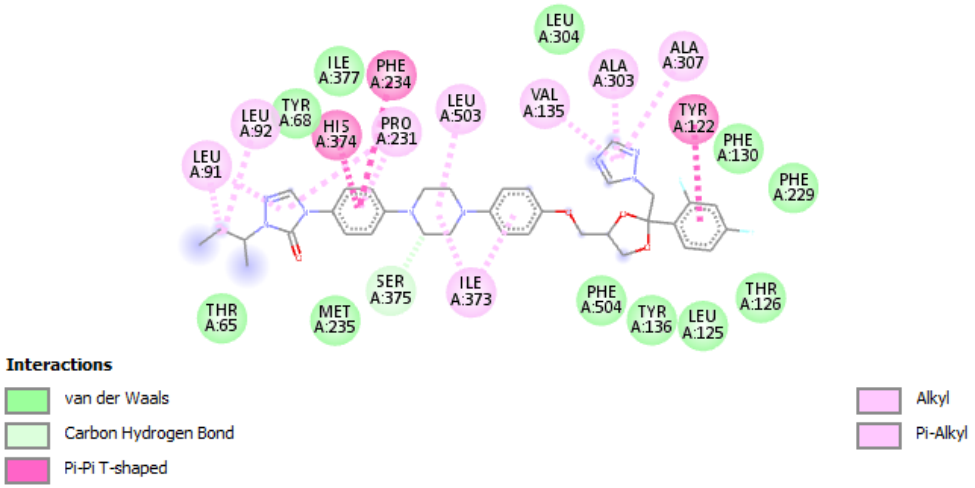

Figure 3. Molecular interactions between *saperconazole* and *CYP51B*

- ***Stigmasterol* Toxicity**

The physicochemical properties (Table 3) of *stigmasterol* and its predicted LD50 (890mg/kg) which places it in predicted toxicity class IV.

Table 3. Physicochemical properties of *stigmasterol* and its predicted LD_{50}

Property	Value
Molecular Weight	412.69
Hydrogen acceptors	1
Hydrogen donors	1
Molecular refractivity	132.76
Topological polar surface area	20.23
logP	7.8
Predicted LD_{50}	890mg/kg
Predicted Toxicity	IV

Class I: fatal if swallowed (LD50 ≤ 5)

Class II: fatal if swallowed (5 < LD50 ≤ 50)

Class III: toxic if swallowed (50 < LD50 ≤ 300)

Class IV: harmful if swallowed (300 < LD50 ≤ 2000)

Class V: may be harmful if swallowed (2000 < LD50 ≤ 5000)

Class VI: non-toxic (LD50 > 5000)

Predicted LD_{50} accuracy = 70.97%

4. Discussion

Current antifungal treatments rely on synthetic fungicides, which raise concerns regarding resistance, environmental impact, and toxicity. Natural compounds from *Eucalyptus* leaf extract, particularly *Stigmasterol*, have shown promise as potential antifungal agents. *Stigmasterol* is a natural compound that has shown potential pharmacological relevance, its likely role as a modulator of the *CYP51B* enzyme which is critical in the sterol biosynthesis pathway of pathogenic fungi is supported by its predicted interactions with the enzyme. The *CYP51B* enzyme, a *lanosterol 14α-demethylase*, plays an indispensable role in converting *lanosterol* into *ergosterol*, a primary component of fungal cell membranes, essential for maintaining membrane fluidity, integrity, and function. Targeting *CYP51B* with inhibitors has been an effective strategy in antifungal therapy, exemplified by azole drugs like *saperconazole* and pramiconazole. These drugs bind to the active site of *CYP51B*, disrupting the fungal sterol biosynthesis pathway, leading to compromised cell membrane stability and, ultimately, fungal cell death. (Zhang et al., 2019).

Stigmasterol's binding energy relative to *saperconazole* and *pramiconazole* is promising, as lower binding energy typically correlates with greater binding affinity, suggesting that *Stigmasterol* may exhibit antifungal efficacy similar to synthetic azole drugs. The competitive binding profile of *Stigmasterol* may also indicate its potential as an antifungal lead compound, particularly valuable given the increasing prevalence of resistance to conventional antifungal agents. Studies have highlighted the role of *CYP51* mutations in conferring resistance to azole drugs, a growing concern in managing fungal infections, especially in immunocompromised populations (Berger et al., 2017). The use of natural compounds like *Stigmasterol* may provide an alternative approach with a distinct binding mechanism, potentially bypassing common resistance pathways associated with synthetic drugs.

The molecular interactions observed in this study underline the structural compatibility between *Stigmasterol* and the *CYP51B* binding site. The hydrogen bond between *Stigmasterol* and Serine 375 is indicative of a strong, conventional hydrogen bond. Serine residues are known for their role in stabilising ligands within active sites. This bond could facilitate a high degree of stability and specificity within the *CYP51B* active site, reinforcing *Stigmasterol's* potential as a *CYP51B* modulator. Moreover, the carbon-hydrogen bond with *Histidine* 374 complements the hydrogen bonding, offering additional stabilization to *Stigmasterol* within the binding site. *Histidine* residues, due to their imidazole side chains, are frequently involved in stabilizing ligand orientation, particularly in proteins with hydrophobic and aromatic binding pockets.

The hydrophobic interactions observed with residues *Alanine* 307, *Isoleucine* 373, and *Leucine* 503 reveal an efficient exploitation of the hydrophobic regions within the *CYP51B* binding pocket. Hydrophobic interactions are known to contribute significantly to the overall binding energy and are crucial for the orientation and affinity of hydrophobic ligands in enzyme active sites. The presence of these hydrophobic contacts with non-polar residues suggests that *Stigmasterol's* molecular structure is well-suited to occupying the hydrophobic channels within *CYP51B*, likely contributing to its competitive binding affinity.

Additionally, the Pi-alkyl interactions involving aromatic residues, such as *Tyrosine* 122, *Tyrosine* 136, and *Phenylalanine* 504, are of particular importance. Pi-alkyl interactions are a type of van der Waals forces that enable aromatic rings to interact with non-polar alkyl groups, enhancing ligand binding through favourable entropic contributions. The presence of these interactions between *Stigmasterol* and the aromatic residues in *CYP51B* may further anchor the ligand, improving the specificity and stability of binding.

These molecular insights have broad implications for the design of new antifungal agents. *Stigmasterol's* interaction profile, combining hydrogen bonds with specific amino acids and extensive hydrophobic contacts, provides a molecular framework that could guide structural modifications aimed at optimizing binding affinity. In silico and in vitro studies on *Stigmasterol* derivatives could explore functional groups that enhance hydrogen bonding or hydrophobic contacts, potentially yielding analogs with even greater affinity for *CYP51B* and increased antifungal efficacy. This approach aligns with the growing interest in phytochemicals as templates for drug discovery, especially as they often exhibit multi-target activity, lower toxicity, and reduced potential for resistance development compared to synthetic counterparts.

Moreover, *Stigmasterol's* structural similarities to cholesterol, a major sterol in mammalian cells, raise interesting considerations for its potential effects on host systems, particularly in balancing selectivity between fungal and human *CYP* enzymes. Selectivity studies will be essential in determining the clinical viability of *Stigmasterol* and its derivatives, ensuring efficacy against fungal *CYP51B* without significant off-target effects on human CYP enzymes, particularly those involved in steroidogenesis and xenobiotic metabolism.

5. Conclusion

The findings of this study highlight the potential of *Stigmasterol* as a promising modulator of the *CYP51B* enzyme, a critical target in fungal sterol biosynthesis. Its predicted binding interactions, comparable to those of established azole antifungal drugs, suggest that *Stigmasterol* may exhibit significant antifungal efficacy. The hydrogen bonding with Serine 375, along with additional stabilizing interactions involving *Histidine* 374, Alanine 307, *Isoleucine* 373, and *Leucine* 503, underscores its strong affinity for the *CYP51B* active site. Furthermore, π-alkyl interactions with aromatic residues enhance its binding stability, reinforcing its role as a viable lead compound in antifungal drug development. Given the rising concern of fungal resistance to conventional azoles, *Stigmasterol's* distinct molecular binding profile may provide an alternative approach to circumvent existing resistance mechanisms. Its natural origin and structural compatibility with the *CYP51B* enzyme make it a compelling candidate for further investigation. Future research should focus on optimizing its antifungal activity through structural modifications, ensuring enhanced selectivity for fungal *CYP51B* enzymes to minimize potential off-target effects.

6. Future Recommendations

1) Further in vitro and in vivo studies should be conducted to validate the antifungal efficacy of Stigmasterol against toxigenic fungi affecting sorghum.

2) Structure-activity relationship (SAR) studies are recommended to identify key functional groups responsible for binding affinity and to optimize the molecule for enhanced antifungal activity.

3) Structural modifications of Stigmasterol should be explored to increase selectivity toward fungal CYP51B over human homologs, minimizing potential side effects.

4) Comparative docking studies with other natural and synthetic antifungal compounds could help benchmark Stigmasterol's efficacy and binding profile.

5) Toxicological profiling and ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) analysis should be carried out to assess the safety and drug-likeness of Stigmasterol.

6) Development of formulation strategies (e.g., nanoformulations or encapsulation) to enhance the bioavailability and delivery of Stigmasterol in plant or clinical systems.

Declarations

Source of Funding

This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Competing Interests Statement

The authors declare that they have no competing interests related to this work.

Consent for publication

The authors declare that they consented to the publication of this study.

Authors' contributions

All the authors took part in literature review, analysis, and manuscript writing equally.

Availability of data and materials

Authors are willing to share data and material on request.

Informed Consent

Not applicable for this study.

References

Bandyopadhyay, R., Ortega-Beltran, A., Akande, A., Mutegi, C., Atehnkeng, J., Kaptoge, L., & Cotty, P.J. (2016). Biological control of aflatoxins in Africa: Current status and potential challenges in the face of climate change. World Mycotoxin Journal, 9(5): 771–789. https://doi.org/10.3920/wmj2016.2130.

Banerjee, P., Kemmler, E., Dunkel, M., & Preissner, R. (2024). ProTox 3.0: A webserver for the prediction of toxicity of chemicals. Nucleic Acids Research, 52(w1): w513–w520. https://doi.org/10.1093/nar/gkae303.

Becher, R., & Wirsel, S.G. (2012). Fungal cytochrome P450 sterol 14α-demethylase (CYP51) and azole resistance in plant and human pathogens. Applied Microbiology and Biotechnology, 95(4): 825–840. https://doi.org/10.1007/s00253-012-4195-9

Berman, H.M., Westbrook, J., Feng, Z., Gilliland, G., Bhat, T.N., Weissig, H., Shindyalov, I.N., & Bourne, P.E. (2000). The Protein Data Bank. Nucleic Acids Research, 28(1): 235–242. https://doi.org/10.1093/nar/28.1.235.

BIOVIA (2020). Discovery Studio Visualizer (Version v21.1.0.20298) [Computer software]. Dassault Systèmes. https://discover.3ds.com/discovery-studio-visualizer-download (Accessed: 21 December 2024).

Dallakyan, S., & Olson, A.J. (2015). Small-molecule library screening by docking with PyRx. Methods in Molecular Biology (Clifton, N.J.), 1263: 243–250. https://doi.org/10.1007/978-1-4939-2269-7_19.

Djouahri, A., Boudarene, L., Meklati, B.Y., Nasri, F., & Dicko, A. (2013). In vitro antimicrobial activity of essential oils and methanolic extracts of *Eucalyptus camaldulensis* Dehnh. Industrial Crops and Products, 45: 155–162. https://doi.org/10.5812/jjnpp.65050.

Forli, S., Huey, R., Pique, M.E., Sanner, M.F., Goodsell, D.S., & Olson, A.J. (2016). Computational protein-ligand docking and virtual drug screening with the AutoDock suite. Nature Protocols, 11(5): 905–919. https://doi.org/10.1038/nprot.2016.051

Haussmann, B.I., Rattunde, H.F., Weltzien-Rattunde, E., Traoré, P.S., Vom Brocke, K., & Parzies, H.K. (2012). Breeding strategies for adaptation of pearl millet and sorghum to climate variability and change in West Africa. Journal of Agronomy and Crop Science, 198(5): 327–339. https://doi.org/10.1111/j.1439-037x.2012.00526.x

Kim, S., Thiessen, P.A., Bolton, E.E., Chen, J., Fu, G., Gindulyte, A., Han, L., He, J., He, S., Shoemaker, B.A., Wang, J., Yu, B., Zhang, J., & Bryant, S.H. (2016). PubChem Substance and Compound databases. Nucleic Acids Research, 44: d1202–d1213. https://doi.org/10.1093/nar/gkv951.

Morris, G.M., Huey, R., Lindstrom, W., Sanner, M.F., Belew, R.K., Goodsell, D.S., & Olson, A.J. (2009). AutoDock4 and AutoDockTools4: Automated docking with selective receptor flexibility. Journal of Computational Chemistry, 30(16): 2785–2791. https://doi.org/10.1002/jcc.21256.

Pettersen, E.F., Goddard, T.D., Huang, C.C., Couch, G.S., Greenblatt, D.M., Meng, E.C., & Ferrin, T.E. (2004). UCSF Chimera—A visualization system for exploratory research and analysis. Journal of Computational Chemistry, 25(13): 1605–1612. https://doi.org/10.1002/jcc.20084.

Pitt, J.I., Hocking, A.D., Glenn, D.R., & Vicentini, M.C. (2013). Mycotoxin production and toxigenic fungi in Australia. Food Control, 34(2): 568–576. https://doi.org/10.1016/j.ijfoodmicro.2006.02.005.

Watkins, R., Wu, L., Zhang, C., & Davis, R.M. (2010). The fungal CYP51s: Their functions, structures, related drug targets, and prospects for antifungal resistance. Biochimica et Biophysica Acta (BBA)-Proteins and Proteomics, 1804(3): 595–604. https://doi.org/10.3389/fmicb.2019.00691.