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Molecular Docking Assessment of Environmental Pesticides as Potential Endocrine Disruptors of Human Estrogen Receptor Alpha

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Research Article

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Molecular Docking Assessment of Environmental Pesticides as Potential Endocrine Disruptors of Human Estrogen Receptor Alpha

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Abstract

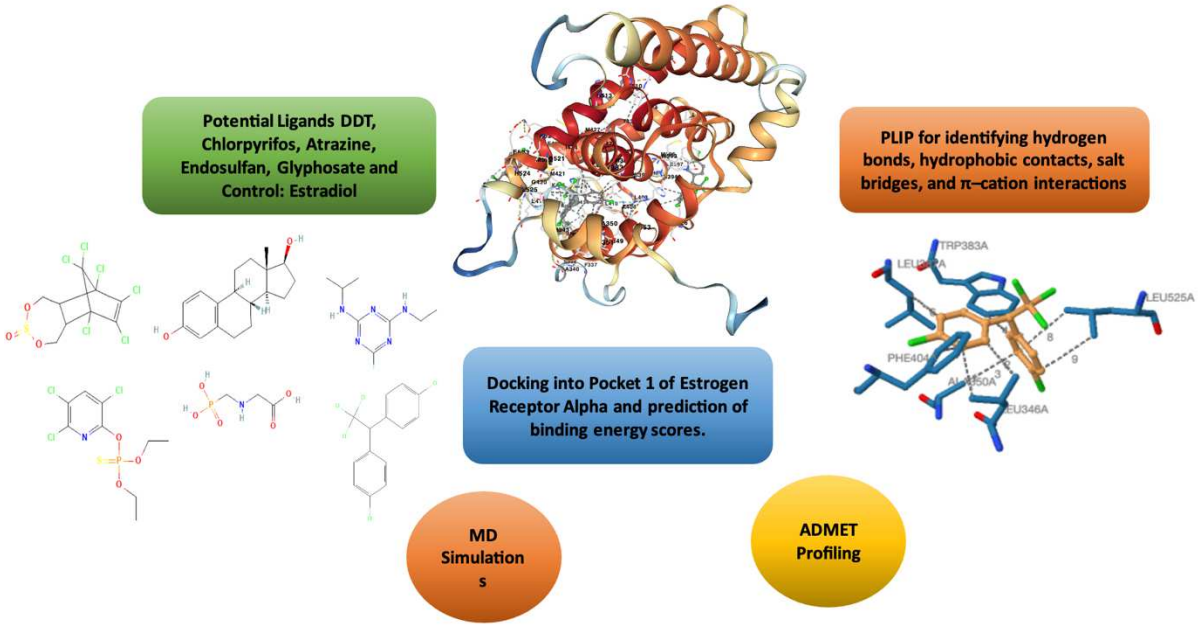
Environmental pesticides are increasingly recognized as potential endocrine-disrupting chemicals (EDCs) due to their ability to mimic or block hormone action. This study investigates the binding affinities and molecular interactions of five commonly used pesticides — DDT, Chlorpyrifos, Endosulfan, Atrazine, and Glyphosate — with the human Estrogen Receptor Alpha (ERα) using in silico methods. The natural ligand Estradiol was docked as a control to validate the binding site. Docking simulations were performed using CB-Dock, and binding interactions were analysed via the PLIP server. DDT showed a strong binding affinity (-8.5 kcal/mol), close to Estradiol (-9.3 kcal/mol), forming extensive hydrophobic contacts. Chlorpyrifos and Endosulfan demonstrated moderate binding (-6.3 and -6.0 kcal/mol, respectively), while Atrazine showed mixed interactions (-5.9 kcal/mol). Glyphosate exhibited the weakest binding (-4.7 kcal/mol) with mainly polar contacts. These results suggest that certain pesticides can bind competitively within the natural hormone pocket of ERα, potentially disrupting estrogenic signaling. This computational study highlights the utility of molecular

docking as a predictive screening tool for EDC risk assessment and emphasizes the need for further experimental validation.

Keywords

Pesticides; Endocrine-disrupting chemicals; Estrogen Receptor Alpha; Molecular docking; CB-Dock; PLIP, MD Simulations, ADMET

Graphical Abstract



1. Introduction

Crop yields intended for human consumption are significantly threatened by a wide range of pests. Research suggests that pest-related losses can range from around 50% to more than 80% of total crop production (Oerke., 2006). As a result, pest and disease management strategies have long played a crucial role in enhancing agricultural productivity worldwide. In parallel, the global burden of malaria has been rising in various regions. Data indicate that in tropical countries, the prevalence of malaria among pregnant women lies between 10% and 20%, while among children, it ranges from 4.7% to 49.7% (Kirui., 2024). To address this, the World Health Organization recommends the widespread use of insecticide-treated bed nets. Pesticides—including herbicides, insecticides, fungicides, and rodenticides—are commonly employed in both agricultural and public health settings. Various chemical classes of pesticides are available, such as pyrethroids, organophosphates, phenyl pyrazoles, benzoylphenyl urea, methylenedioxyphenyl derivatives, and organochlorines. These compounds are used to control a broad spectrum of pests including weeds, microbial pathogens, and animal pests, and they are also used in the treatment of parasitic infections like scabies and diseases caused by lice and mites.

Studies have shown a significant rise in per capita pesticide use in South America, particularly in Brazil, where pesticide consumption surged by 700% between 1985 and 2015 (Tariq and Mushtaq., 2023). Across various parts of Africa, pesticides are widely used not only in agriculture but also for the control of disease vectors such as tsetse flies, houseflies, mosquitoes, and ticks. Similarly, numerous countries in Asia rely on pesticides extensively for agricultural purposes. Widespread use of pesticides in agriculture and healthcare has led to routine, direct or indirect exposure of both humans and animals. While these chemicals are essential for managing pests and enhancing crop yields, growing evidence highlights their harmful effects due to their

inherent toxicity. Although pesticides are formulated to target specific pests, there is a considerable risk of unintended impacts on non-target species. India, notably, has emerged as the world's largest pesticide consumer (Subash et al., 2017). As a result, frequent exposure—whether occupational, accidental, or environmental—has been linked to various human health issues such as skin and eye irritation, headaches, and nausea. In more severe cases, both acute and chronic toxicities have been observed due to pesticide contact. While existing literature reports a range of adverse effects on vital organs, there is still a lack of comprehensive understanding of the molecular mechanisms through which pesticides impair male reproductive health (Gautam et al., 2024). Pesticide exposure can impair brain function by interfering with the regulation of thyroid hormones. These hormones—primarily thyroxine (T4) and triiodothyronine (T3)—are essential for managing metabolism, energy balance, and the normal functioning of the nervous and cardiovascular systems. Certain chlorinated hydrocarbons, such as Dichloro-Diphenyl-Trichloroethane (DDT), have been found to disrupt thyroid hormone levels, potentially causing hypothyroidism (Kosarek & Preston., 2024). This condition may manifest in the form of neurological symptoms including fatigue, paranoia, and depressive behaviours. Cardiovascular toxicity is another significant concern associated with pesticide exposure. Numerous studies have reported that pesticides can lead to abnormalities in heart function, and in extreme cases, even fatal outcomes (Adeyemi et al., 2021). Organophosphate pesticides like chlorpyrifos (CPF), methyl parathion, and dichlorvos are linked to disruptions in autonomic balance—initially stimulating sympathetic activity, followed by extended parasympathetic dominance (Gill et al., 2010) (Barnett., 2023). These disturbances can result in ECG irregularities, such as QT interval prolongation, which may lead to life-threatening arrhythmias like torsade de pointes, and subsequent

myocardial injury. The liver, a primary organ for detoxification, is also vulnerable to damage from toxic substances, including pesticides. Hepatotoxicity refers to liver injury caused by chemicals, which can impair its function(Neuman., 2020). Pesticides are known to contribute to liver damage by inducing oxidative stress and the production of reactive oxygen species (ROS), particularly in the case of organophosphates like chlorpyrifos (Sule et al., 2022) .Pulmonary toxicity, which involves damage to lung tissues, is another potential outcome of pesticide exposure. This can compromise respiratory function and contribute to the development of chronic lung diseases. Alpha-cypermethrin, for example, has been associated with pulmonary fibrosis in experimental studies. It increases oxidative stress in lung tissue, elevates hydroxyproline levels—a marker of fibrosis—and enhances inflammatory responses, as indicated by cytokine activity (Soliman et al., 2021).

Endocrine-disrupting chemicals (EDCs), including many pesticides, can negatively impact reproductive health in both males and females (Ghosh et al., 2022). These chemicals can alter hormonal balance, disrupt normal ovarian function, damage follicles, and ultimately lead to reduced fertility. Certain organochlorine compounds—such as methoxychlor, chlordecone, dieldrin, endosulfan, and dichlorvos—have been shown to interfere with the estrous cycle in rodents and reduce the number of corpora lutea, which are essential for producing progesterone to sustain the uterine lining and early stages of pregnancy (Venkidasamy et al., 2021). Male reproductive health is similarly affected by pesticide exposure. Fertility in males, defined as the ability to fertilize a female partner, can be compromised by environmental toxins. Pesticides can harm male reproductive organs and germ cells, interfering with spermatogenesis and hormonal regulation. Since these organs are responsible for the proliferation and

maturation of sperm cells, any toxic insult can severely impair male fertility (Moreira et al., 2021).

The endocrine system is a complex network of glands responsible for producing and releasing hormones that regulate numerous physiological processes in the body. This finely tuned system can be disrupted by certain substances known as endocrine-disrupting chemicals (EDCs), which interfere with hormonal balance and function in both humans and animals, leading to various health complications. Pesticides are among the chemicals known to behave like EDCs, as they can mimic or block normal hormonal activity, ultimately disrupting the functions of multiple organ systems. Research has linked pesticide exposure to a range of health issues, including skin and eye irritation, acute cardiovascular and neurological conditions, respiratory problems, congenital abnormalities, cancers, and disorders of the kidneys and liver—some of which can be fatal (Ali et al., 2020). Ingestion of specific pesticides, such as chlorpyrifos (CPF) and permethrin, has been associated with gastrointestinal symptoms like vomiting, abdominal pain, diarrhea, mucosal irritation, and more severe effects such as headaches, unconsciousness, sore throat, and skin lesions. Additionally, pesticides have been shown to negatively affect reproductive organs, leading to reduced fertility in both humans and animals. The overall toxicological impact of pesticides is broad, affecting various body systems. Pesticides such as DDT, Chlorpyrifos, and Atrazine have long been suspected EDCs due to their persistence and bioaccumulation (Chaturvedi et al., 2023). The Estrogen Receptor Alpha (ERα) is a critical target for assessing potential estrogenic disruption(Borgert et al., 2024). **(Fig 1).** This study aims to evaluate the binding affinities and interaction profiles of five environmental pesticides with ERα, comparing them to Estradiol as the natural ligand.

Fig. 1. 2D Structures of potential endocrine disruptors (EDCs) and control Estradiol

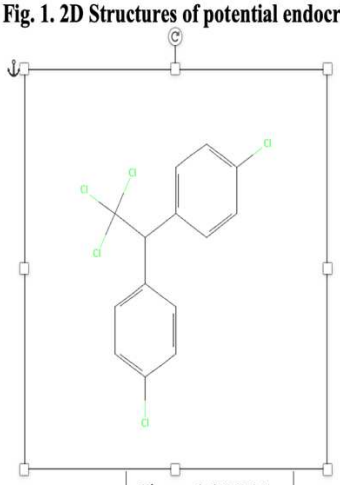


Figure 1.1 DDT



Figure 1.2 Atrazine

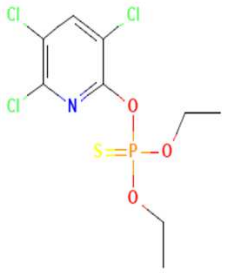


Figure 1.3 Chloropyrifos

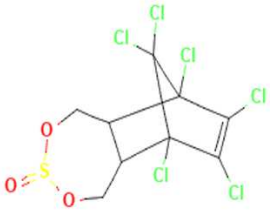


Figure 1.4 Endosulfan

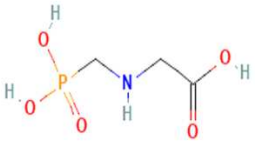


Figure 1.5 Glyphosate

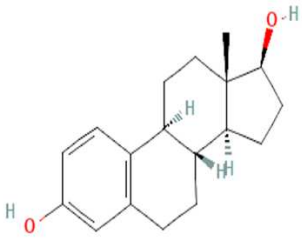


Figure 1.6 Estradiol

2. Materials and Methods

2.1 Protein Preparation

The 3D structure of the human estrogen receptor alpha (ERα) ligand-binding domain was retrieved from the Protein Data Bank (PDB ID: 3ERT). The structure was selected based on its resolution and co-crystallization with a native ligand to ensure docking accuracy.

2.2 Ligand Preparation

Five widely used environmental pesticides known for potential endocrine-disrupting effects were selected as ligands: Atrazine (CID: 2256); DDT (CID: 3036); Chlorpyrifos (CID: 2730); Endosulfan (CID: 32263); Glyphosate (CID: 3496) and Estradiol (CID: 5757). 3D conformers for each compound were downloaded from PubChem in .sdf format and validated for 3D geometry.

2.3 Docking Protocol

Molecular docking was performed using the CB-Dock web server which uses Auto Dock Vina for pose prediction and binding energy calculation (Liu et al., 2020). Protein and ligand files were uploaded, and the tool automatically detected potential binding cavities and performed docking into the top five predicted pockets shown in **Fig. 2.** Estradiol was docked to validate the natural binding pocket. For each ligand, the pose with the lowest binding energy was selected for analysis (Maruthanila et al., 2019).

Fig 2. Molecular docking of potential Endocrine disruptors (EDCs) with Estrogen Receptor Alpha (Erα)

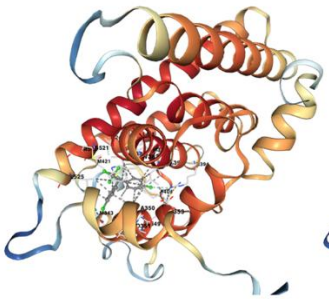


Figure 2.1 DDT

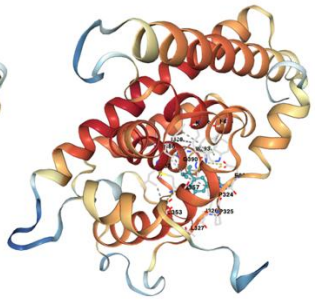


Figure 2.2 Atrazine

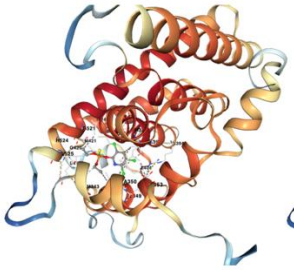


Figure 2.3 Chloropyrifos

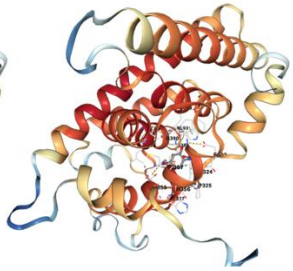


Figure 2.4 Endosulfan

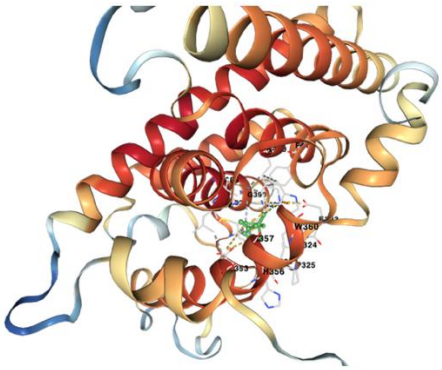


Figure 2.5 Glyphosate

2.4 Interaction Analysis

PLIP (Protein–Ligand Interaction Profiler) was used to identify hydrogen bonds, hydrophobic contacts, salt bridges, and π–cation interactions (Bolz et al., 2025).

3. Results

All ligands were found to dock preferentially into Pocket 1, corresponding to the natural hormone-binding site of the estrogen receptor. DDT exhibited the strongest binding affinity, suggesting a high potential for estrogen mimicry or disruption. Docking results for the five pesticides revealed their potential to bind within the main ligand-binding cavity of ERα. **Table 1.** summarizes the binding affinities:

Table 1: Molecular docking summary of potential Endocrine disruptors (EDCs) with Estrogen Receptor Alpha (Erα)

Pesticide	Binding Score (kcal/mol)	Binding Pocket	Interpretation
DDT	-8.5	Pocket 1	Strong binding; likely endocrine disruptor
Chlorpyrifos	-6.3	Pocket 1	Moderate to strong binding
Endosulfan	-6.0	Pocket 1	Moderate binding
Atrazine	-5.9	Pocket 1	Moderate binding
Glyphosate	-4.7	Pocket 1	Weak binding; less likely disruptor

DDT is makes multiple strong hydrophobic interactions with nonpolar residues in the receptor's hormone-binding domain. Chlorpyrifos, like DDT, mainly uses hydrophobic contacts to stabilize within the receptor's ligand-binding pocket. Residues LEU346 and LEU391 overlap with some of DDT's contact sites, suggesting they are critical for van der Waals stabilization.ILE424 adds additional non-polar interactions deeper into the pocket. Atrazine shows a mixed binding mode,

forming multiple hydrogen bonds plus limited hydrophobic interactions. Notably, GLU323 and LYS449 are recurrent hydrogen-bonding partners — these residues also appear in Endosulfan and Glyphosate binding, suggesting they're important for polar contacts within the binding pocket. Hydrophobic contacts with PRO324 and ILE386 add moderate stabilization but are fewer compared to DDT. Endosulfan engages the receptor through a mixed binding mode: Hydrophobic residues (ILE326, MET357) stabilize the molecule within the binding pocket. Hydrogen bonds with GLU323, ARG394, and LYS449 provide additional polar anchoring. ARG394 also forms a π–cation interaction, suggesting a strong electrostatic attraction with aromatic regions of the ligand. Glyphosate, unlike DDT and Chlorpyrifos, does not form hydrophobic interactions, but instead stabilizes in the pocket mainly through polar interactions. Its 3 hydrogen bonds and salt bridge with LYS449 help anchor it within the receptor's ligand-binding domain. GLU353, ARG394, and LYS449 are all highly polar residues lining the hormone-binding cavity, often involved in recognizing endogenous ligands like estradiol. Endosulfan shows a total of 3 hydrogen bonds + 2 hydrophobic + 1 π–cation interaction Main residues: GLU323, ARG394, LYS449, ILE326, MET357 Mixed binding mode: polar contacts + hydrophobic + π–cation shown in **Fig 3.** The interaction summary is given in **Table 2.**

Fig 3. Protein Interaction Ligand Profiler Analysis

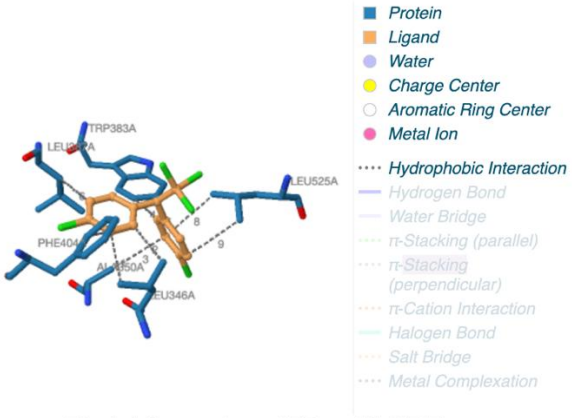


Fig 3.1 Interaction of ERα with DDT

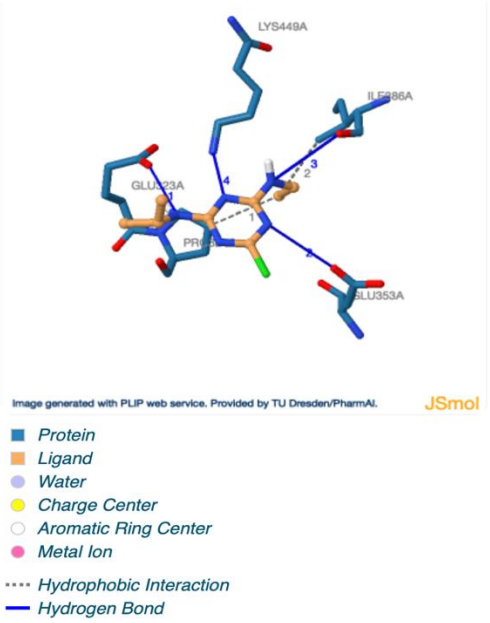


Fig 3.2 Interaction of ERα with Atrazine

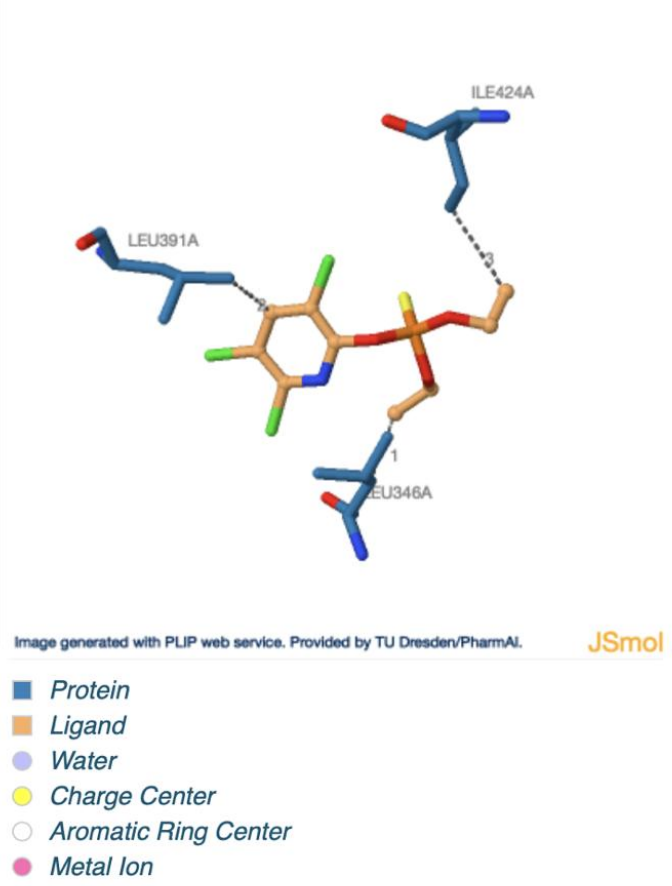


Fig 3.3 Interaction of ERα with Chloropyrifos

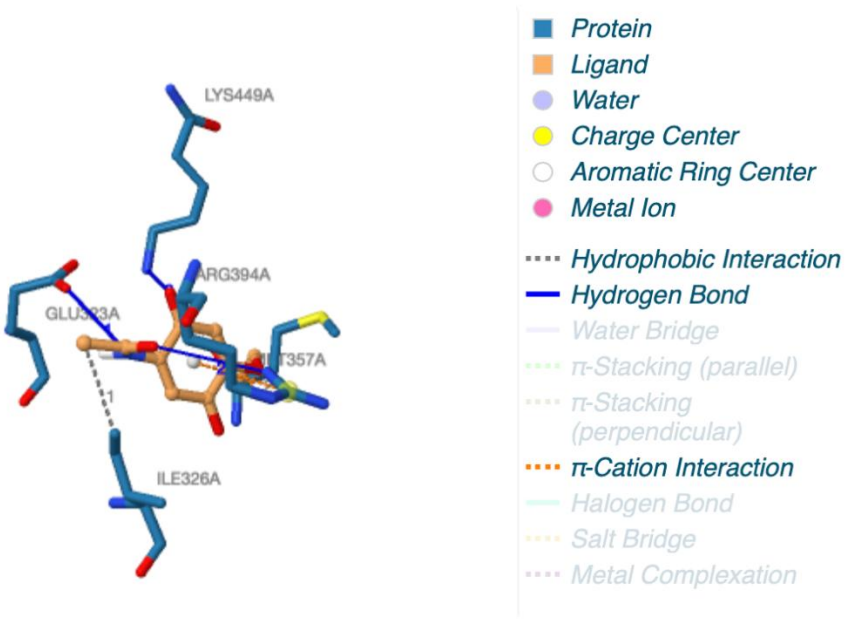


Fig 3.4 Interaction of ERα with Endosulfan

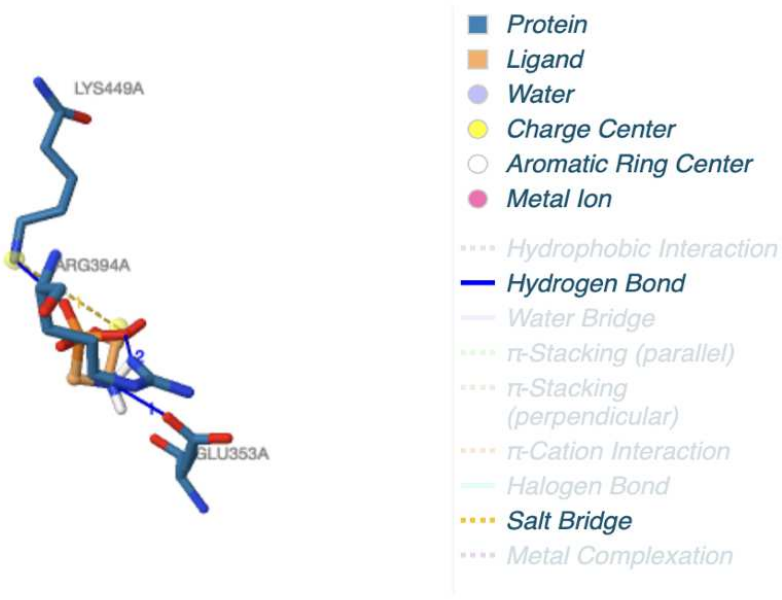


Fig 3.5 Interaction of ERα with Glyphosate

Table 2: Table 2. Protein ligand Interaction summary of potential Endocrine disruptors (EDCs) with Estrogen Receptor Alpha (Erα)

Ligand	Binding Score (kcal/mol)	Hydrogen Bonds	Hydrophobic Contacts	Salt Bridge	π– Cation	Key Residues Involved
DDT	-8.5	0	6	0	0	LEU346, ALA350, TRP383, LEU387, PHE404, LEU525
Chlorpyrifos	-6.3	0	3	0	0	LEU346, LEU391, ILE424
Endosulfan	-6.0	3	2	0	1	GLU323, ARG394, LYS449, ILE326, MET357
Atrazine	-5.9	4	2	0	0	GLU323, GLU353, ILE386, LYS449, PRO324
Glyphosate	-4.7	3	0	1	0	GLU353, ARG394, LYS449

4. Discussions

4.1 Molecular Docking Analysis

Molecular docking analysis identified Pocket 1 of the Estrogen Receptor as the common and most favorable binding site for all tested compounds, including the natural hormone Estradiol and the five pesticides: DDT, Chlorpyrifos, Endosulfan, Atrazine, and Glyphosate. Pocket 1, which has the largest cavity volume (1983 A^0), is confirmed as the receptor's natural ligand-binding domain, based on Estradiol's high binding affinity of -9.3 kcal/mol. Among the pesticides, DDT showed the strongest binding (-8.5 kcal/mol), closely approaching that of Estradiol and indicating a high potential to mimic natural estrogen signaling. Chlorpyrifos (-6.3 kcal/mol) and Endosulfan (-6.0 kcal/mol) demonstrated moderate binding affinities, suggesting they may also competitively interact with the receptor, albeit less potently. Atrazine (-5.9 kcal/mol) showed slightly weaker but still biologically relevant interaction. In contrast, Glyphosate had the weakest binding energy (-4.7 kcal/mol), suggesting a minimal likelihood of direct receptor disruption. These results support the hypothesis that pesticides with binding energies stronger than -6.0 kcal/mol may act as potential endocrine-disrupting chemicals (EDCs) by mimicking or interfering with estrogen signaling at the receptor level (Mazurek., 2024). Docking the native ligand Estradiol yielded a binding energy of -9.3 kcal/mol, confirming Pocket 1 as the hormone-binding site. DDT, with a binding score of -8.5 kcal/mol, shows comparable affinity, suggesting a high potential to compete with natural estrogens for receptor occupancy. This reinforces the risk of DDT acting as an endocrine disruptor. These findings support

existing toxicological data and provide molecular-level evidence that can guide future experimental validation and risk assessment. The study highlights the importance of computational tools like molecular docking in early screening of environmental toxicants for hormonal interference potential.

4.2 **Protein Ligand Interaction Profiler (PLIP) Analysis**

PLIP analysis of the DDT–ERα complex revealed extensive hydrophobic interactions involving key residues such as LEU346, TRP383, and PHE404. These contacts likely contribute to the strong binding affinity observed in molecular docking (-8.5 kcal/mol), indicating potential for estrogen receptor modulation. Chlorpyrifos binds by forming a compact hydrophobic cluster, which aligns with its moderate binding score. This supports its classification as a potential endocrine disruptor due to its ability to occupy the estrogen receptor's hormone-binding cavity. Atrazine likely anchors to ERα mainly via polar hydrogen bonds rather than deep hydrophobic packing. This moderate interaction pattern aligns with its classification as a possible but weaker EDC compared to strong binders like DDT. Endosulfan's multiple interaction types indicate it likely binds more flexibly than purely hydrophobic ligands. This makes it capable of potential estrogen receptor modulation, consistent with its known classification as an EDC. Glyphosate relies purely on hydrogen bonding and ionic interactions, but lacks the hydrophobic fit necessary for strong receptor activation or disruption. This molecular evidence suggests it is less likely to function as a direct EDC via ERα binding compared to the other pesticides. Atrazine forms four hydrogen bonds and two hydrophobic contacts within the ERα binding pocket, primarily involving residues GLU323, GLU353, ILE386, and LYS449. Endosulfan exhibits a similar mixed interaction mode with three hydrogen bonds, two hydrophobic interactions, and a

notable π–cation interaction with ARG394, indicating a stable, flexible binding pattern consistent with moderate affinity (Hazarika et al., 2020).

4.3 MD Simulations an ADMET Prediction

Due to server limitations, full-scale MD simulations could not be performed. Future work should include long-term MD studies to validate the stability of pesticide–ERα complexes under dynamic conditions. While in silico ADMET prediction could not be performed due to server unavailability, literature data **(Table 3)** clearly shows that DDT and Endosulfan exhibit high lipophilicity (logP > 4) and bioaccumulation potential, supporting their persistence as environmental EDCs (Chaturvedi et al., 2023) .In contrast, Glyphosate shows poor bioaccumulation due to its low logP, which aligns with its weak binding to ERα observed in docking (Hutton., 2022).

Table 3. Toxicity summary of potential EDCs with Reported logP values from PubChem and EPA assessments

Pesticide	logP	Bioaccumulation	Known Toxicity	Reference
DDT	~6.9	High	Probable human carcinogen (IARC)	Tasselli et al., 2023
Chlorpyrifos	~4.7	Moderate	Neurotoxic, possible developmental toxin	Banjare et al., 2021

Endosulfan	~4.7	High	Banned, reproductive toxin	Bhanot & Pant (2025)
Atrazine	~2.6	Low–Moderate	Possible endocrine disruptor, carcinogen	Perez et al., 2022
Glyphosate	~-3.2	Negligible	Controversial; probable carcinogen (IARC)*	Li & Fantke et al., (2023)

*These values are approximate “Reported logP values from PubChem and EPA assessments.”

5. Conclusion

This in silico study demonstrates that multiple environmental pesticides, particularly DDT, exhibit strong binding affinities with Estrogen Receptor Alpha (ERα) and interact within the same hormone-binding pocket as the native ligand Estradiol. DDT showed a binding score of -8.5 kcal/mol, close to Estradiol’s -9.3 kcal/mol, supported by extensive hydrophobic interactions with key residues such as LEU346, TRP383, and PHE404. Other pesticides, including Chlorpyrifos and Endosulfan, displayed moderate binding energies (-6.3 and -6.0 kcal/mol respectively) with mixed hydrophobic and polar interactions, suggesting potential endocrine-disrupting effects. Glyphosate’s weak binding (-4.7 kcal/mol) indicates a low probability of direct receptor-mediated disruption. These findings provide molecular-level evidence supporting the classification of certain pesticides as potential endocrine disruptors.

6. Future Prospects

While the molecular docking and interaction analyses provide valuable preliminary insights, further validation using long-term Molecular Dynamics (MD) simulations could clarify the dynamic stability of these complexes. Additionally, comprehensive ADMET profiling and wet-lab experiments, such as receptor binding assays and gene expression studies, are recommended to confirm the in silico predictions and assess the real-world impact of pesticide exposure on hormonal pathways. This integrated approach will enhance the understanding of pesticide-induced endocrine disruption and support better regulatory and risk assessment frameworks.

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