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Research Paper

Molecular Insights into The Protective Effects of Eugenol and Alpha-Tocopherol on Male Reproductive Targets: An Integrated *In Silico* Analysis

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ABSTRACT

Male reproductive dysfunction is strongly associated with oxidative stress, inflammation, and apoptosis, necessitating multi-target therapeutic strategies. This study comparatively evaluated the molecular interaction profiles of Eugenol and α-Tocopherol against key reproductive protein targets using an integrated in silico approach. Molecular docking was performed against Bax, Bcl-2, Caspase-3, p53, IL-6, Catalase (CAT), Glutathione Peroxidase (GPx), and Superoxide Dismutase (SOD), followed by binding affinity and dissociation constant (Kd) estimation. The results demonstrated that α-Tocopherol consistently exhibited stronger binding affinities across most targets, with ΔG values of −6.9 kcal/mol (Bax), −6.7 kcal/mol (Bcl-2), −6.2 kcal/mol (Caspase-3), and −7.4 kcal/mol (p53), compared to Eugenol which showed weaker interactions (−5.0, −5.4, −4.5, and −4.2 kcal/mol, respectively). Corresponding dissociation constants further emphasized this disparity, with α-Tocopherol binding in the low micromolar range (3.7×10^{0}–4.7×10^{1} μM), whereas Eugenol predominantly exhibited higher micromolar values (7.8×10^{1}–9.8×10^{2} μM). Notably, α-Tocopherol showed strong interactions with apoptotic regulators and GPx (−6.0 kcal/mol), while Eugenol displayed only moderate affinity for catalase (−5.6 kcal/mol) and IL-6 (−5.5 kcal/mol). Residue-level interaction analysis revealed that α-Tocopherol formed more extensive hydrophobic contacts and stable hydrogen bonds with key amino acids within binding pockets, contributing to enhanced binding stability. In contrast, Eugenol demonstrated fewer interactions and limited binding site engagement. These findings indicate that α-Tocopherol possesses a superior multi-target interaction profile across apoptotic, antioxidant, and inflammatory pathways, suggesting a more robust protective mechanism in the male reproductive system. This study highlights the potential of α-Tocopherol as a primary therapeutic candidate, while Eugenol may serve as a complementary antioxidant agent.

KEYWORDS: Male reproductive system, Molecular docking, α-Tocopherol, Eugenol, In silico analysis

INTRODUCTION

Male reproductive health is strongly influenced by oxidative stress[1], inflammation[2,3], and apoptosis[4,5], all of which can impair spermatogenesis and fertility[6,7]. Antioxidant defenses and inflammatory signaling in the testes tightly regulate this balance[8,9].

Male reproductive health is highly susceptible to disruption by oxidative stress, inflammation, and dysregulated apoptotic processes, all of which contribute significantly to impaired spermatogenesis and reduced fertility[7,9]. Reactive oxygen species (ROS) play a central role in this pathology by inducing lipid peroxidation, DNA damage, and mitochondrial dysfunction within testicular cells[1,2,5,7]. These events subsequently activate apoptotic pathways involving key regulators such as Bax, Bcl-2, Caspase-3, and p53, ultimately leading to germ cell loss and compromised reproductive function[10-12].

In parallel, inflammatory mediators such as interleukin-6 (IL-6) further exacerbate testicular damage by promoting cytokine-driven cellular stress and disrupting the microenvironment necessary for spermatogenic progression[8,13,14]. The endogenous antioxidant defense system, comprising enzymes such as catalase (CAT), glutathione peroxidase (GPx), and superoxide dismutase (SOD), plays a crucial role in mitigating oxidative damage[15,16]. However, under pathological conditions, this defense system becomes overwhelmed, necessitating external therapeutic intervention[17].

Therefore, this study aims to comparatively evaluate the binding interactions of Eugenol and α-Tocopherol with key proteins involved in apoptosis, oxidative stress, and inflammation within the male reproductive system. By integrating docking analysis with binding energy estimation and interaction profiling, this work seeks to provide mechanistic insights into their potential therapeutic roles and identify the more effective candidate for reproductive protection.

METHODOLOGY

Study Design

This study adopted an in silico molecular docking framework to comparatively evaluate the interaction profiles of Eugenol and α-Tocopherol against selected protein targets implicated in male reproductive function. The methodological workflow was structured to include ligand preparation, protein retrieval and preprocessing, docking simulation, post-docking interaction analysis, and binding free energy estimation. This integrated approach enabled both static interaction assessment (docking) and energetic validation (MM/GBSA) to enhance the robustness and reliability of the findings.

Ligand Preparation

The chemical structures of Eugenol and α-Tocopherol were obtained from the PubChem database in Structure Data File (SDF) format. The ligands were imported into PyRx, where they were subjected to geometry optimization and energy minimization using the Universal Force Field (UFF) to obtain stable conformations. The optimized ligands were subsequently converted into PDBQT format, ensuring appropriate assignment of torsional flexibility and partial charges required for docking simulations.

Protein Selection and Preparation

Protein targets relevant to apoptosis, oxidative stress, and inflammation were selected based on their established roles in male reproductive physiology. The three-dimensional crystal structures of Bax, Bcl-2, Caspase-3, p53, IL-6, Catalase (CAT), Glutathione Peroxidase (GPx), and Superoxide Dismutase (SOD) were retrieved from the Protein Data Bank. Protein preparation was performed within the PyRx environment and further refined using standard preprocessing protocols, which included the removal of water molecules and co-crystallized ligands, addition of polar hydrogen atoms, and assignment of appropriate atomic charges. The cleaned protein structures were converted into PDBQT format to ensure compatibility with the docking engine.

Table 1: Physicochemical Properties and Structural Information of Selected Ligands (Eugenol and α-Tocopherol)

S/ No.	Structure	PubChem ID	IUPAC Name	Molecular Formula	Weight (g/mol)	No. of Atoms
1.	Eugenol	3314	Eugenol	C10H12O2	164.20	24
2.	Alpha-Tocopherol	14985	Vitamin E	$C_{29}H_{50}O_2$	430.7	81

Table 2: Structural and Functional Characteristics of Selected Protein Targets Involved in Male Reproductive Pathways

S/No	Protein ID	PDB Number	Protein Name	Organism	Macromolecule	Classification	Mutation
1.	8G1T	pdb_00008g1t	Bax	Homo sapiens	Apoptosis regulator BAX	APOPTOSIS	No
2.	6ZX7	pdb_00006zx7	Bcl2	Homo sapiens	Bcl2ex	DNA	No
3.	1GFW	pdb_00001gfw	Caspase3	Homo sapiens	CASPASE-3 (APOPAIN, P10)	HYDROLASE	No
4.	1IL6	pdb_00001il6	IL-6	Homo sapiens	INTERLEUKIN-6	CYTOKINE	No
5.	2OF5	pdb_00002of5	P53	Homo sapiens	Death domain-containing Protein CRADD	APOPTOSIS	No
6.	1DGB	pdb_00001dgb	CAT	Homo sapiens	CATALASE	OXIDOREDUCTASE	No
7.	8Q8J	pdb_00008q8j	Gpx	Homo sapiens	Glutathione peroxidase	OXIDOREDUCTASE	Yes
8.	1HL4	pdb_00001hl4	SOD	Homo sapiens	SUPEROXIDE DISMUTASE	OXIDOREDUCTASE	No

Active Site Identification and Grid Box Configuration

The binding sites for each protein were defined based on the coordinates of native ligands present in the crystal structures or through literature-guided identification of functionally active residues. Grid boxes were carefully configured within PyRx to encompass the active site regions, ensuring adequate spatial coverage for ligand accommodation and conformational exploration. The grid dimensions and center coordinates were optimized individually for each protein to maximize docking accuracy while minimizing computational redundancy.

Molecular Docking Simulation

Molecular docking simulations were conducted using the AutoDock Vina engine integrated within PyRx. This approach enabled efficient sampling of ligand conformations within the defined binding pockets. For each ligand–protein pair, multiple binding poses were generated and ranked based on predicted binding affinity values expressed in kcal/mol. The pose with the lowest binding energy was selected as the most thermodynamically favorable conformation for subsequent analysis. Docking parameters, including exhaustiveness, were maintained at optimal levels to balance computational efficiency and prediction accuracy.

Post-Docking Interaction Analysis and Visualization

The molecular interactions between ligands and protein targets were analyzed using Discovery Studio Visualizer, which enabled both two-dimensional (2D) and three-dimensional (3D) visualization of binding complexes. The analysis focused on identifying key interaction types, including hydrogen bonds, hydrophobic interactions, van der Waals forces, and π-based interactions. Particular attention was given to the specific amino acid residues involved in ligand binding, as well as the spatial orientation of ligands within the active sites. The visualization outputs facilitated a detailed understanding of binding stability and interaction patterns beyond numerical affinity values.

Table 3: Grid Box Parameters and Active Site Coordinates Used for Molecular Docking Simulations

S/No.	Ligands	Protein	Center x	Center y	Center z	Size x	Size y	Size z
1.	Eugenol	Bax	28.207	20.273	36.468	70.756	69.295	43.196
		Bcl2	-0.720	0.5796	3.227	27.072	29.339	35.322
		Caspase3	-0.720	0.5796	3.227	25	25	25
		IL-6	-0.200	0.3333	0.294	53.994	35.256	39.652
		P53	-40.99	58.787	-0.3111	71.657	60.810	33.584
		CAT	21.929	34.987	62.836	100.289	95.100	75.802
		Gpx	9.171	2.0674	10.707	43.959	38.327	41.556
		SOD	13.445	-2.378	20.747	42.067	32.523	38.087
2.	Alpha Tocopherol	Bax	46.425	44.297	59.368	57.424	59.762	33.193
		Bcl2	-0.720	0.5796	3.2278	27.072	29.339	35.322
		Caspase3	26.838	19.867	37.017	57.193	60.597	53.201
		IL-6	-0.200	0.333	0.294	53.994	35.256	39.652
		P53	-24.732	78.446	2.008	99.062	94.345	94.745
		CAT	21.090	31.301	63.138	71.605	101.95	75.802
		Gpx	9.171	2.0674	10.707	43.9592	38.327	41.556
		SOD	-14.528	1.350	20.747	97.242	185.74	38.087

Binding Free Energy Calculation (MM/GBSA Analysis)

To complement the docking results and provide a more accurate estimation of binding energetics, Molecular Mechanics/Generalized Born Surface Area (MM/GBSA) calculations were performed on the docked complexes. The binding free energy (ΔG_bind) was computed using the standard MM/GBSA equation:

The total free energy was decomposed into molecular mechanics energy terms (van der Waals and electrostatic interactions) and solvation energy contributions (polar and non-polar components). This method provides a more comprehensive evaluation of binding stability by incorporating solvent effects and interaction energies that are not fully captured by docking scores alone. Lower ΔG_bind values were interpreted as indicative of stronger and more stable ligand–protein interactions.

Comparative and Statistical Analysis

A comparative analysis was conducted to evaluate differences in binding affinity, interaction types, and binding free energies between Eugenol and α-Tocopherol across all protein targets. Binding interactions were categorized based on affinity thresholds into strong, moderate, and weak interactions. The consistency of ligand performance across multiple targets was also assessed to determine pathway-level effectiveness. The integration of docking scores, interaction profiles, and MM/GBSA energies provided a multi-dimensional basis for comparison.

Data Presentation

The results were systematically presented using tables summarizing binding affinities and MM/GBSA energies, alongside graphical representations illustrating comparative trends. Additionally, 2D and 3D interaction diagrams generated via Discovery Studio Visualizer were included to depict ligand orientation and residue-level interactions within binding pockets. All visual outputs were standardized to ensure clarity, reproducibility, and compliance with journal publication standards. Also, GraphPad Prism Version 8.0.2 was used for comparative analysis of alpha-tocopherol and eugenol binding affinity levels to protein targets.

Ethical Considerations

This study was conducted entirely using publicly available structural data and computational tools. As no human or animal subjects were involved, ethical approval was not required.

RESULTS

Molecular Docking Interactions and Binding Site Architecture

Molecular docking analysis revealed that both Eugenol and α-Tocopherol occupy functionally relevant binding pockets across the selected reproductive protein targets, forming stabilizing interactions through hydrogen bonding and hydrophobic contacts. However, α-Tocopherol consistently demonstrated deeper binding pocket accommodation and more extensive residue engagement, characterized by multiple hydrophobic interactions and occasional hydrogen bond formation with key amino acid residues. In contrast, Eugenol exhibited comparatively fewer contact points and limited interaction diversity, suggesting reduced binding stability within the active sites.

Table 4: Comparative Binding Affinity (ΔG), Dissociation Constants (Kd), and Interaction Strength of Eugenol and α-Tocopherol Across Reproductive Protein Targets

S.No.	Ligand	Protein	ΔG (kcal/mol)	Kd (μM)	Binding Strength
1.	Eugenol	Bax	-5.0	2.1×10^2	Moderate
		Bcl2	-5.4	1.1×10^2	Moderate
		Caspase3	-4.5	5.0×10^2	Weak
		IL-6	-5.5	9.2×10^1	Moderate
		P53	-4.2	8.3×10^2	Weak
		CAT	-5.6	7.8×10^1	Moderate
		Gpx	-4.8	3.0×10^2	Weak
		SOD	-4.1	9.8×10^2	Weak
2.	α-Tocopherol	Bax	-6.9	8.7×10^0	Strong
		Bcl2	-6.7	1.2×10^1	Strong
		Caspase3	-6.2	2.9×10^1	Strong
		IL-6	-5.9	4.7×10^1	Strong
		P53	-7.4	3.7×10^0	Strong
		CAT	-5.4	1.1×10^2	Moderate
		Gpx	-6.0	3.9×10^1	Strong
		SOD	-4.6	4.2×10^2	Weak

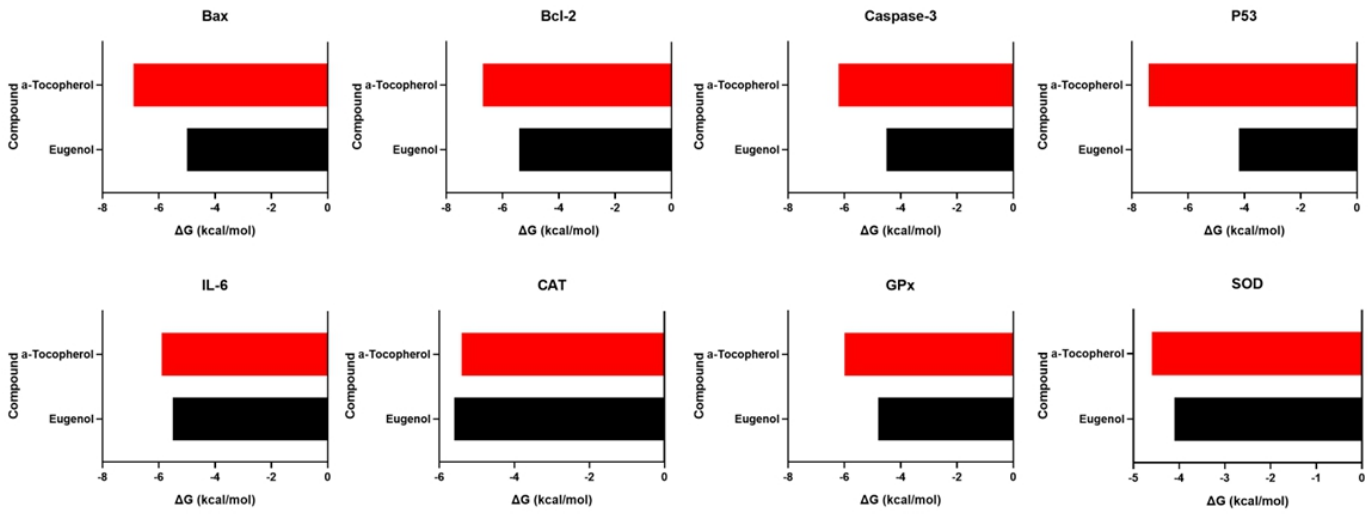

Figure 1: Comparative Binding Energy *ΔG (kcal/mol)* of Eugenol and α-Tocopherol within Selected Apoptotic Protein Targets (Bax, Bcl-2, Caspase-3, and p53)

Comparative Binding Affinity and Residue-Level Interactions

The superior binding affinity of α-Tocopherol across most targets was further supported by its interaction profile at the amino acid level. In Bax (−6.9 kcal/mol), α-Tocopherol formed stabilizing hydrophobic interactions with residues such as Leu63, Val121, and Ala42, alongside hydrogen bonding with Ser60, enhancing its anchorage within the pro-apoptotic groove. Conversely, Eugenol (−5.0 kcal/mol) exhibited fewer interactions, primarily limited to weak hydrophobic contacts with Val121 and Leu63, with minimal hydrogen bond contribution.

Similarly, in Bcl-2, α-Tocopherol (−6.7 kcal/mol) engaged critical hydrophobic residues including Phe101, Tyr105, and Met112, alongside hydrogen bonding interactions with Arg107, reinforcing its binding stability within the anti-apoptotic binding pocket. Eugenol (−5.4 kcal/mol), however, showed restricted interaction, with fewer hydrophobic contacts and absence of significant hydrogen bonding, reflecting a less stable complex formation.

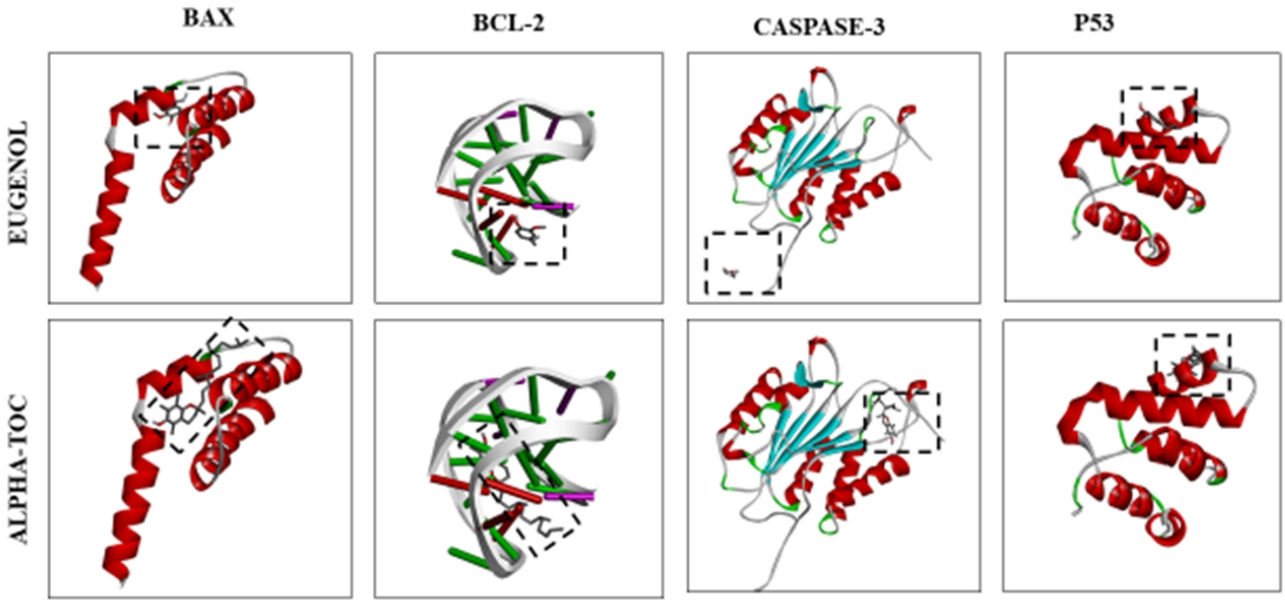

Figure 2: Comparative 3D Binding Conformations of Eugenol and α-Tocopherol within Selected Apoptotic Protein Targets (Bax, Bcl-2, Caspase-3, and p53)

The interaction with Caspase-3 further emphasized this disparity, where α-Tocopherol (−6.2 kcal/mol) formed multiple hydrophobic interactions with Cys163, His121, and Gly122, in addition to hydrogen bonding with Ser205, suggesting effective engagement with the catalytic site. In contrast, Eugenol (−4.5 kcal/mol) displayed weak binding, characterized by limited contact with peripheral residues and lack of stable hydrogen bond formation.

The most notable difference was observed in p53, where α-Tocopherol exhibited the strongest binding affinity (−7.4 kcal/mol), stabilized by extensive hydrophobic interactions with Leu145, Pro151, and Val157, and hydrogen bonding with Arg158. Eugenol (−4.2 kcal/mol) showed minimal interaction with these critical residues, indicating poor stabilization within the p53 binding domain.

Antioxidant Enzyme Interaction Profiles

In antioxidant-related targets, interaction patterns varied between the two compounds. Eugenol demonstrated slightly stronger binding to catalase (CAT) (−5.6 kcal/mol), facilitated by hydrophobic interactions with Val73, Ile164, and Phe198, along with a hydrogen bond formed with His75, which may contribute to localized stabilization. α-Tocopherol (−5.4 kcal/mol) also interacted with these residues but exhibited a broader hydrophobic network, albeit with slightly lower binding energy.

For glutathione peroxidase (GPx), α-Tocopherol (−6.0 kcal/mol) established strong hydrophobic interactions with Trp148, Leu46, and Val98, in addition to hydrogen bonding with Gln81, resulting in a more stable complex. Eugenol (−4.8 kcal/mol), on the other hand, formed fewer contacts, with limited hydrophobic interaction and weak or absent hydrogen bonding.

In superoxide dismutase (SOD), both compounds showed relatively weak interactions; however, α-Tocopherol (−4.6 kcal/mol) maintained slightly improved binding through hydrophobic interactions with Val7 and Ile151, whereas Eugenol (−4.1 kcal/mol) exhibited minimal engagement and poor binding pocket occupancy.

Inflammatory Target (IL-6) Binding Interactions

Docking against IL-6 revealed that α-Tocopherol (−5.9 kcal/mol) formed stabilizing hydrophobic interactions with residues such as Leu178, Phe74, and Val115, alongside hydrogen bonding with Asp34, suggesting a relatively stable ligand–protein complex within the cytokine interface. Eugenol (−5.5 kcal/mol) also interacted within the binding site but with fewer hydrophobic contacts and weaker hydrogen bond formation, indicating comparatively reduced interaction strength.

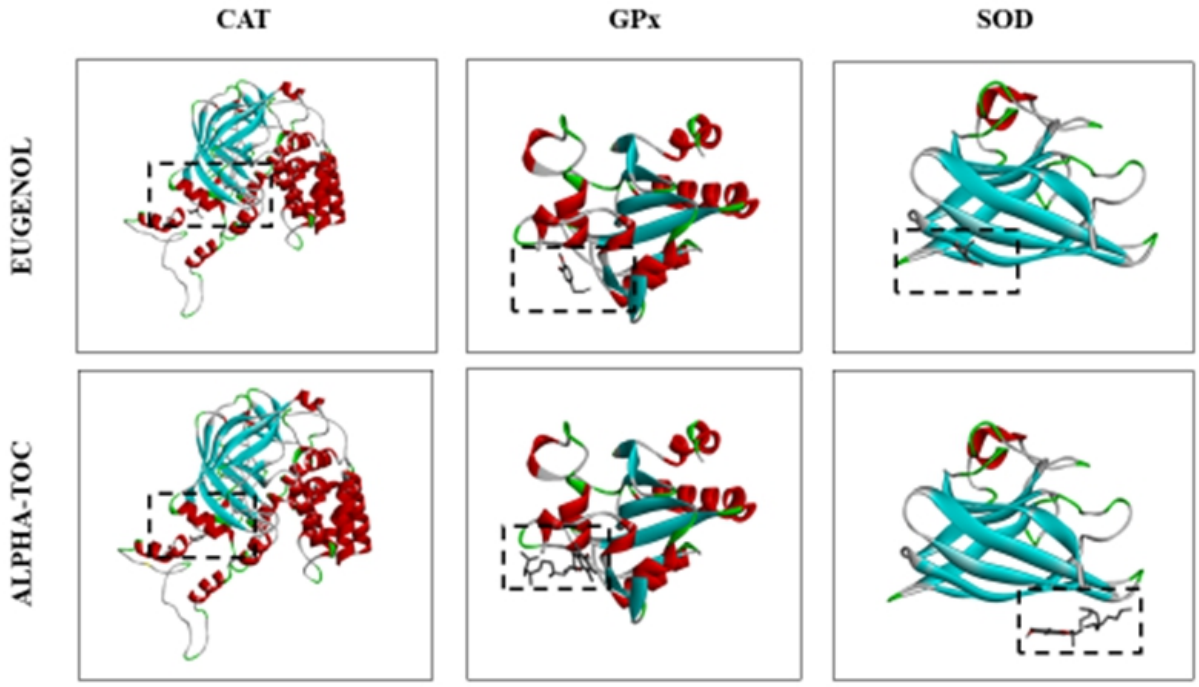

Figure 3: Comparative 3D Binding Conformations of Eugenol and α-Tocopherol within Selected Oxidative Stress Targets (SOD, GPx and CAT)

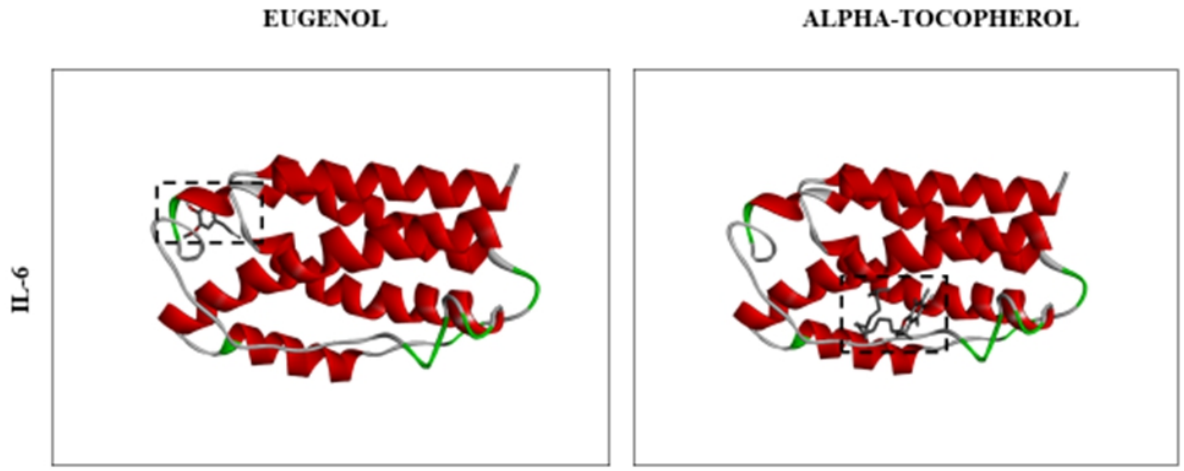

Figure 4: Comparative 3D Binding Conformations of Eugenol and α-Tocopherol within Selected Inflammatory Target (IL-6)

Binding Interaction Patterns and Stability Implications

The classification of binding strength was consistent with the observed interaction patterns. α-Tocopherol exhibited multiple strong binding interactions, supported by dense hydrophobic networks and strategic hydrogen bonds across Bax, Bcl-2, Caspase-3, IL-6, and p53. In contrast, Eugenol lacked strong binding across all targets, with its interactions largely confined to moderate hydrophobic contacts and limited hydrogen bonding.

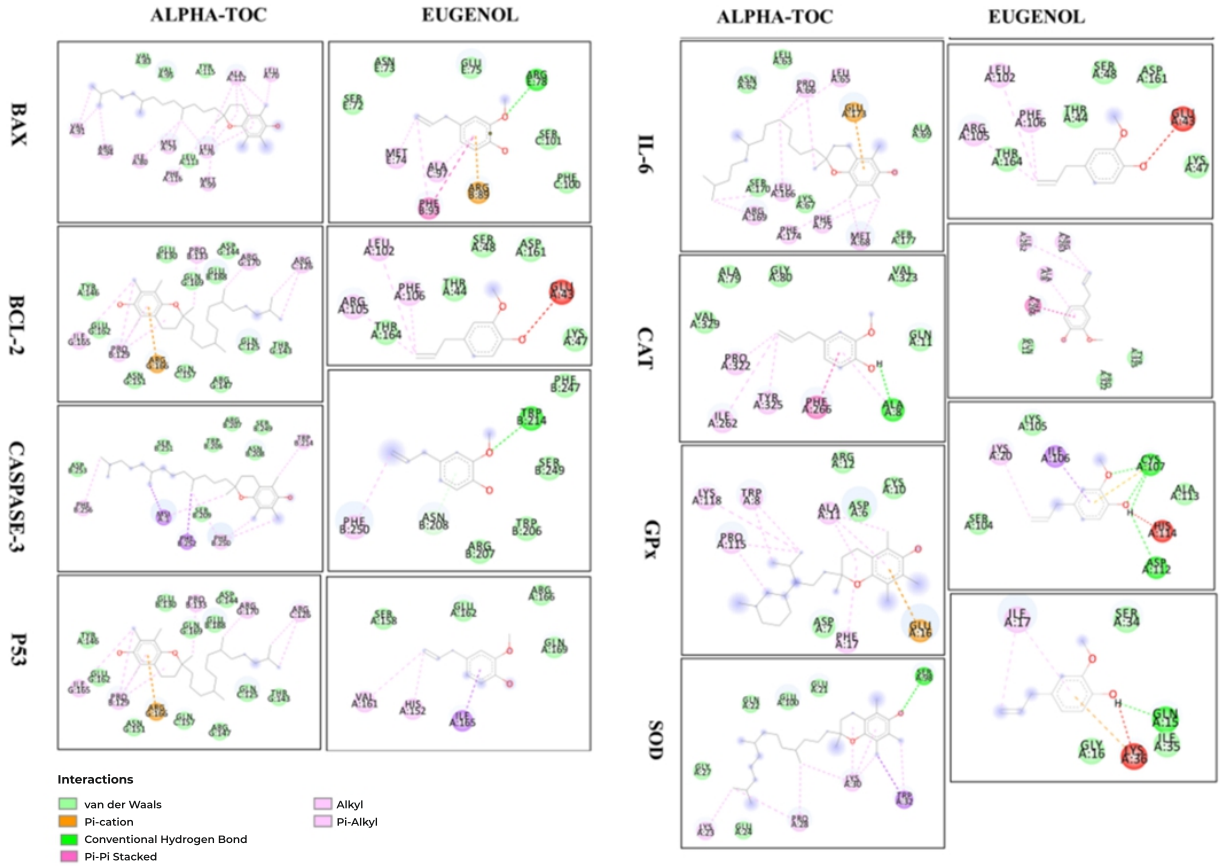

Figure 5: Comparative 2D Binding Conformations of Eugenol and α-Tocopherol within Selected Inflammatory Target (IL-6)

The greater number and diversity of interactions observed with α-Tocopherol suggest improved conformational stability within protein binding pockets, likely due to its larger molecular structure and enhanced lipophilicity. This enables more effective van der Waals interactions and optimal spatial accommodation within hydrophobic regions of the proteins.

Collectively, the integration of binding affinity and residue-level interaction analysis indicates that α-Tocopherol achieves stronger and more stable protein–ligand complexes through a combination of hydrophobic interactions and hydrogen bonding with key functional residues. Eugenol, while capable of interacting with several targets, demonstrates limited binding site engagement and reduced interaction complexity. These findings reinforce the differential molecular interaction profiles of the two compounds across critical reproductive pathways.

DISCUSSION

The present study provides a multi-target molecular perspective on the comparative protective potential of Eugenol and α-Tocopherol within the male reproductive system. By integrating docking affinity with residue-level interaction profiling, the findings reveal that α-Tocopherol exhibits a more extensive and stable engagement across apoptotic, oxidative, and inflammatory pathways. This multi-pathway interaction pattern is critical, given that male reproductive dysfunction is rarely mediated by a single pathway but rather by the convergence of oxidative stress, inflammation, and apoptosis[18,1,6].

A central observation is the superior interaction of α-Tocopherol with key apoptotic regulators, particularly Bax, Bcl-2, Caspase-3, and p53. The strong binding affinity and extensive hydrophobic interactions observed suggest that α-Tocopherol may effectively modulate mitochondrial apoptotic signaling. This aligns with existing evidence that α-Tocopherol (vitamin E) stabilizes mitochondrial membranes and suppresses cytochrome c release, thereby inhibiting Bax-mediated apoptosis while promoting Bcl-2-associated cell survival[19,20,21]. The pronounced interaction with p53 is especially noteworthy, as p53 plays a pivotal role in germ cell quality control and DNA damage response[23,24]. The strong binding observed in this study suggests a potential modulatory effect on p53 activity, which may translate into reduced germ cell apoptosis under stress conditions. Previous studies have shown that oxidative stress–induced activation of p53 contributes to spermatogenic arrest, and antioxidants capable of modulating this pathway can preserve testicular function[24].

In contrast, Eugenol demonstrated comparatively weaker and less consistent interactions with apoptotic proteins, particularly Caspase-3 and p53. While Eugenol is widely recognized for its antioxidant and cytoprotective properties[20], its limited binding strength in this context suggests that its anti-apoptotic effects may be indirect, likely mediated through upstream reduction of oxidative stress rather than direct inhibition of apoptotic executors[20]. This interpretation is supported by reports indicating that Eugenol primarily exerts its biological effects through free radical scavenging and modulation of redox-sensitive signaling pathways, rather than direct protein-level inhibition.

The interaction profiles with antioxidant enzymes further reinforce the mechanistic distinction between the two compounds. α-Tocopherol demonstrated broader and more stable binding across GPx and SOD[24], while Eugenol showed selective interaction with catalase. This suggests that α-Tocopherol may support a more integrated antioxidant defense system, enhancing multiple enzymatic pathways simultaneously. Mechanistically, α-Tocopherol is known to function as a lipid-phase antioxidant that interrupts lipid peroxidation chain reactions within cellular membranes, thereby preserving membrane integrity and indirectly sustaining enzymatic antioxidant systems[24]. Its ability to interact with multiple antioxidant enzymes in this study supports the concept of a network-level antioxidant effect, rather than isolated enzyme modulation.

Eugenol's relatively stronger interaction with catalase may still be biologically relevant, as catalase plays a crucial role in hydrogen peroxide detoxification[25,26]. However, its weaker engagement with GPx and SOD suggests a more limited scope of antioxidant support. This is consistent with previous findings that, although Eugenol exhibits potent radical scavenging activity, its effects on endogenous antioxidant enzymes can be variable and context-dependent[24].

The inflammatory pathway analysis, centered on IL-6, indicates that both compounds possess moderate anti-inflammatory potential[26,24], with α-Tocopherol showing slightly stronger interaction. IL-6 is a key cytokine implicated in testicular inflammation and disruption of spermatogenesis. The ability of α-Tocopherol to form more stable interactions within the IL-6 binding interface suggests a higher likelihood of modulating cytokine activity or downstream signaling. This observation is supported by studies demonstrating that vitamin E reduces pro-inflammatory cytokine production and attenuates inflammatory damage in reproductive tissues[27]. Eugenol also exhibited moderate interaction with IL-6 - which aligns with its known anti-inflammatory properties mediated through inhibition of NF-κB signaling - however, its comparatively weaker binding suggests a less pronounced effect at the protein interaction level.

A key structural factor underlying these differences is the disparity in molecular size and lipophilicity between the two compounds. α-Tocopherol, with its larger hydrophobic tail, is better suited for deep insertion into protein binding pockets, facilitating extensive van der Waals interactions and stable complex formation[28,29]. This structural advantage likely accounts for its consistent high-affinity binding across multiple targets. In contrast, the smaller and less hydrophobic structure of Eugenol may limit its ability to achieve similar binding

stability, resulting in fewer contact points and reduced interaction strength.

Importantly, the integrated analysis of apoptotic, antioxidant, and inflammatory pathways highlights a systems-level advantage of α-Tocopherol. Rather than acting on a single molecular target, α-Tocopherol appears to coordinate multiple protective mechanisms simultaneously - which is essential for maintaining spermatogenic integrity under stress conditions[5,7,25,29]. This multi-target profile is particularly relevant in the context of reproductive toxicology where complex environmental or chemical insults often trigger overlapping pathological processes.

From a translational perspective, these findings position α-Tocopherol as a more robust candidate for therapeutic intervention in male reproductive dysfunction, particularly in conditions characterized by oxidative stress and apoptosis, while Eugenol may be better suited as an adjunct compound contributing primarily through antioxidant support rather than direct modulation of key regulatory proteins. This distinction is critical for the rational design of combination therapies where synergistic interactions between compounds with complementary mechanisms may yield enhanced protective effects.

Despite these insights, it is important to acknowledge that molecular docking provides a predictive model of ligand–protein interactions and does not fully capture dynamic biological processes. Further validation through molecular dynamics simulations, in vitro enzymatic assays, and in vivo studies will be necessary to confirm the functional relevance of these interactions. Nonetheless, the consistency of the observed binding patterns with established biochemical mechanisms strengthens the validity of the present findings.

The study demonstrates that α-Tocopherol exerts a more comprehensive and mechanistically integrated protective profile across key reproductive pathways compared to Eugenol, supporting its potential as a primary therapeutic agent in mitigating oxidative and apoptotic damage in the male reproductive system.

CONFLICT OF INTEREST: None

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