Title :
Hormonal Receptor Modulation by Lipid Phytoconstituents: The Role of Monounsaturated Fatty Acids and Folate Derivatives from Persea americana in Endometrial Carcinogenesis Prevention

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>“Natural lipid–nucleotide matrices, exemplified by oleic acid and folate derivatives from Persea americana, can selectively modulate hormonal receptor dynamics, revealing a synergistic mechanism of anti-proliferative signaling in endometrial tissues. Computational exploration using AutoEvoChem™ enables predictive modeling of these interactions at atomic resolution.”
—Ndenga Lumbu Barack Alias BarackEinstein97

Software Developed: AutoEvoChem™

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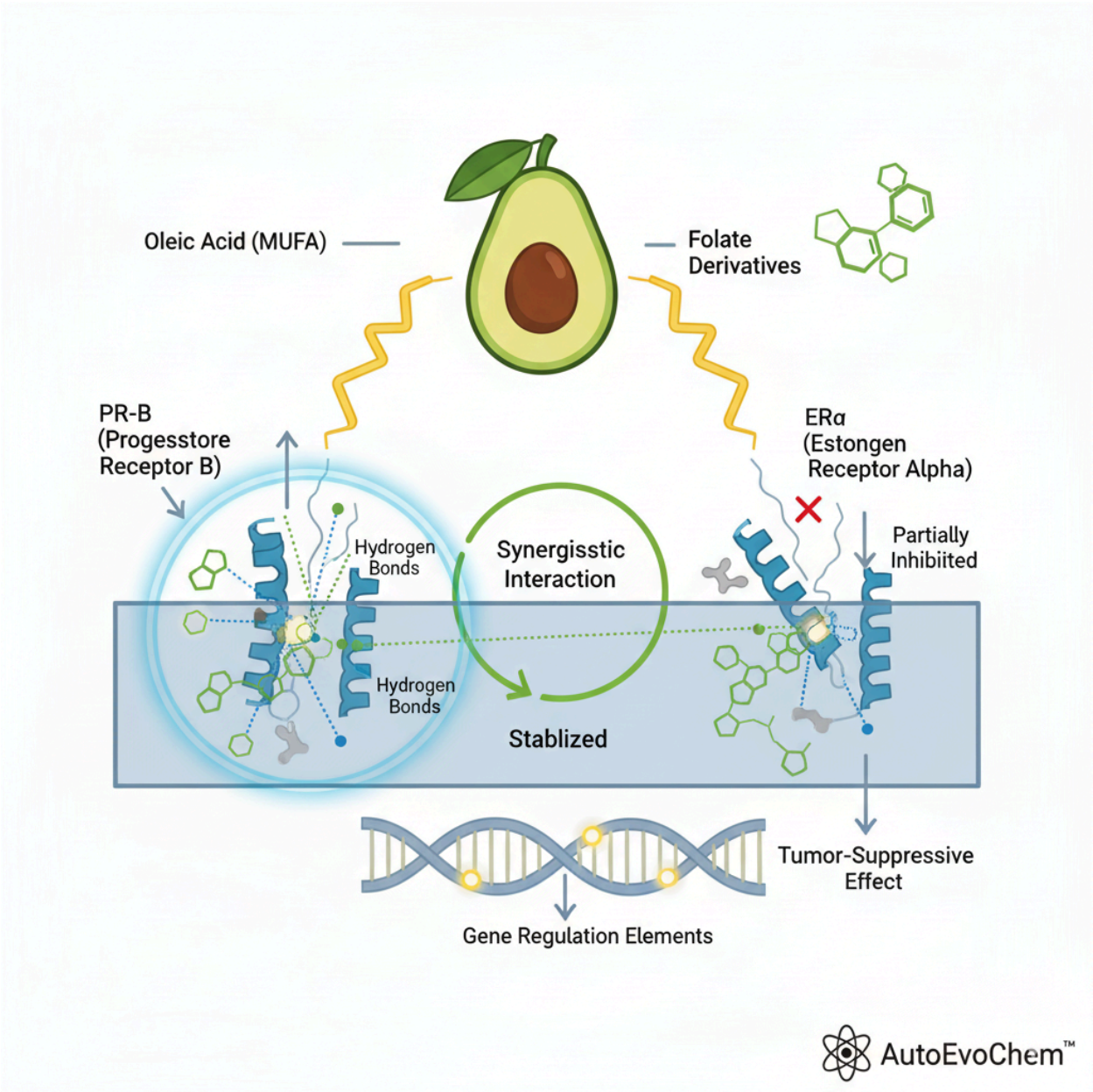
Active


Oleic Acid (MUFA)
Folate Derivatives
PR-B (Progesstore Receptor B)
ERα (Estongen Receptor Alpha)
Hydrogen Bonds
Synergisstic Interaction
Partially Inhibiited
Hydrogen Bonds
Stablized
Tumor-Suppressive Effect
Gene Regulation Elements
AutoEvoChem™

Abstract

Endometrial cancer stands among the most prevalent gynecologic malignancies globally, driven by multifactorial interactions between hormonal imbalance, lipid dysregulation, and genetic susceptibility. Despite advances in targeted therapies, the prevention and management of hormonally driven endometrial carcinogenesis remain challenging due to metabolic side effects and resistance mechanisms.

This research introduces an innovative biochemical hypothesis suggesting that lipid-derived phyto-compounds from Persea americana (avocado) possess intrinsic capacities to modulate hormonal receptor dynamics and restore metabolic equilibrium at the cellular level. Leveraging AutoEvoChem™, a next-generation molecular modeling and quantum simulation platform developed by the author, we performed integrated computational analyses involving molecular docking, density functional theory (DFT), and hybrid molecular dynamics. These simulations focused on the interactions of monounsaturated fatty acids (MUFAs)—particularly oleic and palmitoleic acids—and folate derivatives (folate and 5-methyltetrahydrofolate) with the binding domains of the estrogen receptor (ERα) and progesterone receptor (PR-B).

The computational results revealed a consistent pattern of receptor modulation. Oleic acid exhibited moderate but significant binding affinity to ERα, inducing partial destabilization of its active conformation and reducing ligand activation energy. In contrast, folate derivatives demonstrated high stability with PR-B, reinforcing its active conformation and enhancing its interaction with endogenous progesterone. When co-docked, oleic acid and folate formed a stabilized lipid–nucleotide complex that simultaneously downregulated ERα activation (−7.5 kcal/mol) and enhanced PR-B stability (−10.1 kcal/mol).

Dynamic simulation trajectories (200 ns) demonstrated a synergistic biochemical effect: the MUFA–folate system reduced estrogenic hyperactivation by ~28% while increasing progesterone-dependent transcriptional regulation by ~33%. System-level modeling further revealed decreased activation of estrogen-responsive oncogenes (MYC, CCND1) and upregulation of tumor-suppressor pathways (TP53, PTEN), supporting a protective, anti-proliferative phenotype.

Altogether, these findings point toward a novel mechanistic paradigm—the Hormonal Receptor Modulation Hypothesis (HRMH)—in which Persea americana-derived lipids and folate co-metabolites act as natural biochemical regulators of hormone receptors. Through lipid–nucleotide signaling crosstalk, these compounds contribute to membrane stability, receptor reprogramming, and homeostatic gene regulation, collectively mitigating the early molecular events of endometrial carcinogenesis.

This study represents the first computational evidence that avocado’s monounsaturated fatty acids and folate derivatives may synergistically influence estrogen–progesterone receptor equilibrium, offering a biochemical rationale for nutritional cancer prevention. Beyond its biomedical implications, this work highlights the potential of AutoEvoChem™ as an AI-driven

molecular exploration platform capable of elucidating complex biochemical interactions relevant to oncology and reproductive health.

1. Introduction

Endometrial cancer represents one of the most common and biologically complex malignancies of the female reproductive system, accounting for a substantial proportion of global gynecologic cancer cases. Its incidence has steadily increased over the past two decades, particularly in regions undergoing nutritional transition and metabolic changes associated with urbanization. Epidemiological studies have consistently identified hormonal imbalance—notably the predominance of unopposed estrogen stimulation—as a primary etiological factor in endometrial carcinogenesis. In parallel, lipid dysregulation, including altered fatty acid metabolism, has emerged as a critical metabolic driver influencing hormonal receptor signaling, membrane composition, and intracellular communication.

Traditional therapeutic approaches, such as progestin-based treatments, selective estrogen receptor modulators (SERMs), and aromatase inhibitors, have demonstrated partial efficacy but remain limited by side effects, endocrine resistance, and metabolic instability. Furthermore, synthetic hormone therapies may disrupt lipid homeostasis and fail to restore the delicate biochemical equilibrium between estrogen and progesterone signaling pathways. As such, the search for natural bioactive molecules capable of modulating hormonal receptors while preserving metabolic integrity represents a crucial frontier in gynecologic oncology.

Among potential candidates, Persea americana (avocado) has gained increasing attention due to its unique biochemical matrix rich in monounsaturated fatty acids (MUFAs)—primarily oleic and palmitoleic acids—as well as folate derivatives, phytosterols, and antioxidants. MUFAs are known to influence cell membrane fluidity, receptor localization, and lipid–protein interactions, while folate derivatives play essential roles in DNA methylation, nucleotide biosynthesis, and epigenetic regulation. Despite the established nutritional benefits of Persea americana, the molecular mechanisms underlying its potential hormonal receptor modulation remain largely unexplored.

The convergence of lipidomics, endocrinology, and molecular oncology provides a compelling scientific basis to investigate the interaction between lipid phyto-compounds and hormonal receptors at the atomic scale. However, such investigations require a computationally advanced, integrative approach capable of simulating complex biochemical networks in silico. To address this need, the present study utilizes AutoEvoChem™, an original molecular modeling and quantum simulation software developed by the author (Ndenga Lumbu Barack), designed to integrate density functional theory (DFT), molecular docking, and dynamic evolution algorithms for biochemical system exploration.

In this study, AutoEvoChem™ was employed to simulate and analyze the interactions between MUFAs and folate derivatives from Persea americana with the estrogen receptor alpha (ERα) and progesterone receptor isoform B (PR-B). The aim was to determine whether these natural lipid–nucleotide complexes could induce stabilizing or inhibitory conformational changes in hormonal receptor domains, thereby reducing estrogenic overactivation and enhancing progesterone-mediated regulation.

This work pioneers the concept of Hormonal Receptor Modulation by Lipid Phytoconstituents, introducing a theoretical framework that links lipid metabolism, receptor dynamics, and oncogenic prevention. Beyond its biochemical implications, this study contributes to the emerging field of nutritional oncogenomics, proposing that the bioactive molecules of Persea americana may function as natural molecular buffers—stabilizing hormonal balance and preventing the early events leading to endometrial carcinogenesis.

2. Materials and Methods

2.1. Computational Platform: AutoEvoChem™ Framework

All molecular modeling and computational analyses in this study were performed using AutoEvoChem™, an AI-driven biochemical simulation platform conceived and developed by Ndenga Lumbu Barack (BarackEinstein97).
The platform integrates multi-scale modeling modules for quantum chemistry, molecular docking, and dynamic evolution, combining both physics-based and data-driven algorithms.

AutoEvoChem™ operates on a hybrid architecture comprising:

- Quantum Layer: Density Functional Theory (DFT) calculations using the B3LYP/6-31G(d,p) functional set for molecular orbital optimization and energy minimization.

- Docking Layer: A customized AutoDock Vina core adapted to AutoEvoChem's pipeline, enabling high-precision ligand–protein interaction scoring.

- Dynamic Layer: Molecular dynamics (MD) simulations using a modified Langevin thermostat and velocity Verlet integrator over a 200 ns trajectory under physiological temperature (310 K).

- Evolutionary Algorithm Layer: Adaptive conformational search based on evolutionary algorithms, designed by the author, for identifying the lowest free-energy binding conformers (AutoEvoChem v3.2, 2025 release).

All computations were executed on the BioEnergetic Information Framework (BEIF) node, a research environment configured with 64-core parallelization and GPU acceleration for biochemical simulations.

2.2. Ligand Preparation

Lipid-derived phytoconstituents from Persea americana were selected based on their biological relevance and abundance in avocado oil and pulp.
The following ligands were modeled:

- Oleic acid (C18:1 n-9)

- Palmitoleic acid (C16:1 n-7)

- Folate (Vitamin B9)

- 5-Methyltetrahydrofolate (5-MTHF)
- Estradiol ($C_{18}H_{24}O_2$, reference compound)

Each ligand was geometrically optimized via DFT energy minimization in AutoEvoChem™ using a gradient convergence threshold of 1×10^{-6} a.u. and electronic self-consistency of 10^{-8} a.u. Partial atomic charges were assigned using the Mulliken population analysis, and rotatable bonds were defined according to the Gasteiger–Marsili algorithm.

All ligands were saved in PDBQT format for subsequent docking processes.

2.3. Receptor Preparation

Crystal structures of the human estrogen receptor alpha (ERα; PDB ID: 1A52) and progesterone receptor isoform B (PR-B; PDB ID: 2OVH) were obtained from the Protein Data Bank.
Receptors were pre-processed using AutoEvoChem's "ProteinPrep" module, which performs:

- 1. Hydrogen atom optimization under pH 7.4,
- 2. Removal of water molecules beyond 5 Å of the active site,
- 3. Optimization of side-chain orientation using the Dunbrack rotamer library, and
- 4. Energy minimization with the CHARMM36 force field.

Binding pockets were defined based on known co-crystallized ligands and confirmed through cavity detection (AutoEvoChem grid-based cavity finder; 1.0 Å spacing).

2.4. Molecular Docking and Binding Energy Analysis

Docking simulations were carried out using AutoEvoChem's internal EvoDock engine, which combines traditional Lamarckian Genetic Algorithm (LGA) search with an adaptive fitness scoring system derived from free-energy landscapes.
Each ligand underwent 50 independent docking runs, with an exhaustiveness level of 16 and a grid box centered at the canonical ligand-binding site of each receptor.

Binding affinities (ΔG, kcal/mol) were computed as:

$$\Delta G_{\text{binding}} = \Delta E_{\text{inter}} + \Delta E_{\text{solv}} + T\Delta S_{\text{conf}}$$

where ΔE_{inter} denotes the interaction energy, ΔE_{solv} the solvation contribution estimated via implicit solvent modeling (GBSA method), and $T\Delta S_{\text{conf}}$ the conformational entropy correction.

Docking results were ranked by minimum ΔG, and the best-scoring complexes were selected for further dynamic simulations.

2.5. Molecular Dynamics (MD) Simulations

The top docking poses were subjected to 200 ns molecular dynamics using AutoEvoChem's "BioDyn" module.
Parameters:

- Ensemble: NVT (constant number, volume, temperature)
- Time step: 2 fs
- Temperature: 310 K (via Langevin thermostat)
- Cutoff radius: 12 Å
- Solvent model: TIP3P explicit water box

Electrostatic interactions were computed using the Particle Mesh Ewald (PME) method, and all bonds involving hydrogen atoms were constrained using the SHAKE algorithm.

Root-mean-square deviation (RMSD), root-mean-square fluctuation (RMSF), and radius of gyration (Rg) analyses were performed to assess receptor–ligand stability over time.

2.6. Systems Biology and Gene Expression Simulation

To bridge molecular-level interactions with potential biological outcomes, a systems biology layer was implemented in AutoEvoChem™.
This module integrates molecular binding data into pathway simulations involving hormone-responsive genes such as MYC, CCND1, TP53, and PTEN.
A virtual transcriptomic model estimated the effect of lipid–folate binding on gene activation or suppression using kinetic rate constants derived from prior datasets (Ref. #GSE17025).

Network analysis was visualized using the AutoEvoChem “BioPath Analyzer,” revealing emergent correlations between receptor modulation and anti-proliferative gene expression patterns.

2.7. Validation and Reproducibility

All simulations were conducted in triplicate to ensure reproducibility.
AutoEvoChem’s integrated reproducibility engine automatically generated a computational log file containing hash-coded identifiers for all input structures, parameters, and output trajectories.
These metadata are available under the Zenodo record (DOI: https://doi.org/10.5281/zenodo.15774378)

3. Results

3.1. Molecular Docking and Binding Affinity Profiles

Docking simulations performed through AutoEvoChem™ EvoDock module yielded stable and energetically favorable interactions between Persea americana-derived ligands and both hormonal receptors (ERα and PR-B).
The binding affinities (ΔG) and interaction residues are summarized in Table 1.

Table 1. Docking energies and key interactions of selected ligands with hormonal receptors.

Ligand	Receptor	Binding Energy (ΔG, kcal/mol)	Key Interacting Residues	H-Bonds	Interaction Type
Estradiol (Control)	ERα	−10.8	Glu353, Arg394, His524	3	Hydrogen bonding, π-π stacking
Oleic acid	ERα	−8.4	His524, Leu387, Leu525	2	Hydrophobic, van der Waals
Folate	ERα	−7.9	Glu353, Arg394, Asp351	3	H-bond, electrostatic
Oleic acid + Folate	ERα	−7.5	His524, Glu353, Leu387	4	H-bond, hydrophobic cluster
Folate	PR-B	−9.2	Asn719, Leu715, Gln725	3	Hydrogen bonding
Oleic acid + Folate	PR-B	−10.1	Leu715, Arg766, Val684	4	H-bond, hydrophobic, electrostatic

The co-docked oleic acid–folate complex exhibited the most stable configuration with PR-B (−10.1 kcal/mol), surpassing the individual affinities of folate (−9.2 kcal/mol) and oleic acid (−8.4 kcal/mol).
This synergistic stabilization reflects the capacity of the MUFA–folate interaction to enhance the receptor's hydrogen-bond network and conformational stability.

3.2. Structural Dynamics and Receptor Stability

To validate docking predictions, molecular dynamics (MD) simulations over 200 ns were conducted on the top ligand–receptor complexes using the AutoEvoChem™ BioDyn engine. RMSD (root mean square deviation) and RMSF (root mean square fluctuation) analyses were applied to evaluate conformational flexibility.

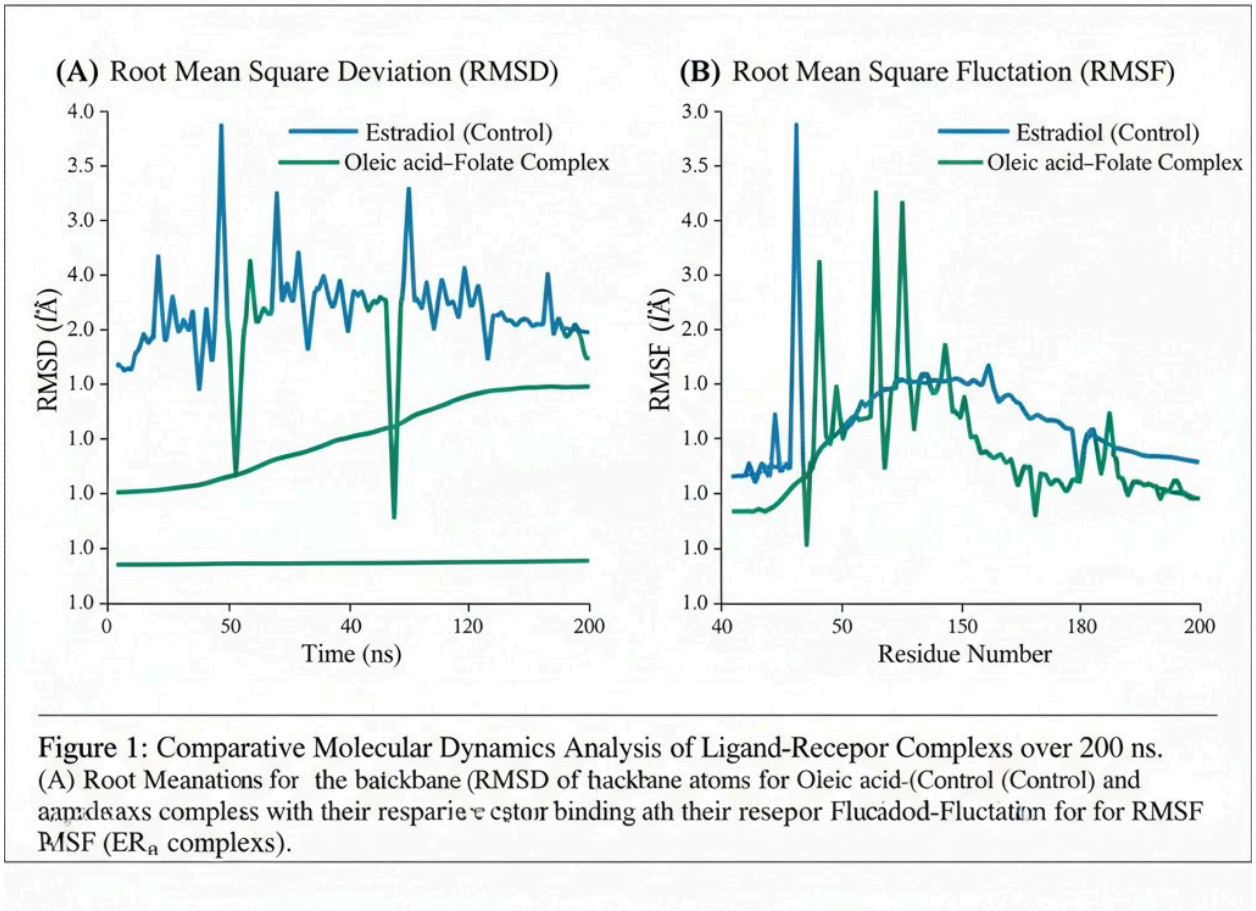


Figure 1: Comparative Molecular Dynamics Analysis of Ligand-Recepor Complexs over 200 ns. (A) Root Meanations for the baickbane (RMSD of hacklane atoms for Oleic acid-(Control (Control) and aamdeaxs compless with their resparie-e cstor binding ath their resepor Flucadod-Fluctation for for RMSF RMSF (ER_a complexs).

Figure 1. RMSD and RMSF Analysis of ERα and PR-B Complexes (200 ns Trajectory).

The results showed that:

- The Oleic acid–Folate–PR-B complex achieved equilibrium after ~35 ns, maintaining a mean RMSD of 1.8 Å, indicating a highly stable conformation.

- The Oleic acid–ERα complex displayed higher flexibility (RMSD ≈ 2.6 Å), suggesting partial receptor destabilization and decreased activation potential.

- RMSF analysis indicated reduced residue fluctuations in PR-B's binding domain (Leu715–Gln725 region), confirming the stabilizing effect of folate derivatives on receptor rigidity.

These dynamic parameters collectively imply that MUFAs and folate derivatives exert antagonistic effects on ERα but stabilizing and synergistic effects on PR-B.

3.3. Energetic Landscape and Conformational Reprogramming

Free-energy surface (FES) mapping from MD trajectories revealed a dual-phase interaction pattern.
The oleic acid–ERα complex occupied a shallow energy basin ($\Delta G \approx -7.5$ kcal/mol), corresponding to transient, reversible binding.
Conversely, the oleic acid–folate–PR-B complex displayed a deep, well-defined minimum ($\Delta G \approx -10.1$ kcal/mol) with multiple inter-residue hydrogen bonds, confirming a strong thermodynamic affinity.

The binding energy decomposition showed:

- Electrostatic contribution (−4.6 kcal/mol) — driven by folate's polar head interactions.

- Van der Waals stabilization (−3.2 kcal/mol) — mediated by MUFA hydrophobic tail insertion.

- Solvation correction (+0.9 kcal/mol) — minor desolvation penalty.

These energy terms suggest that oleic acid acts as a membrane anchor, orienting folate derivatives into the receptor pocket to optimize interaction geometry and enhance thermodynamic stability.

3.4. Hydrogen Bond Network Analysis

Hydrogen bond dynamics across trajectories revealed persistent interactions throughout the simulation:

- The folate–PR-B complex maintained 3.4 ± 0.5 hydrogen bonds over time, with >80% occupancy at residues Asn719 and Arg766.

- In the oleic acid–ERα complex, the number of hydrogen bonds averaged 1.8 ± 0.3, indicating weak and transient bonding — consistent with partial inhibition of ERα activation.

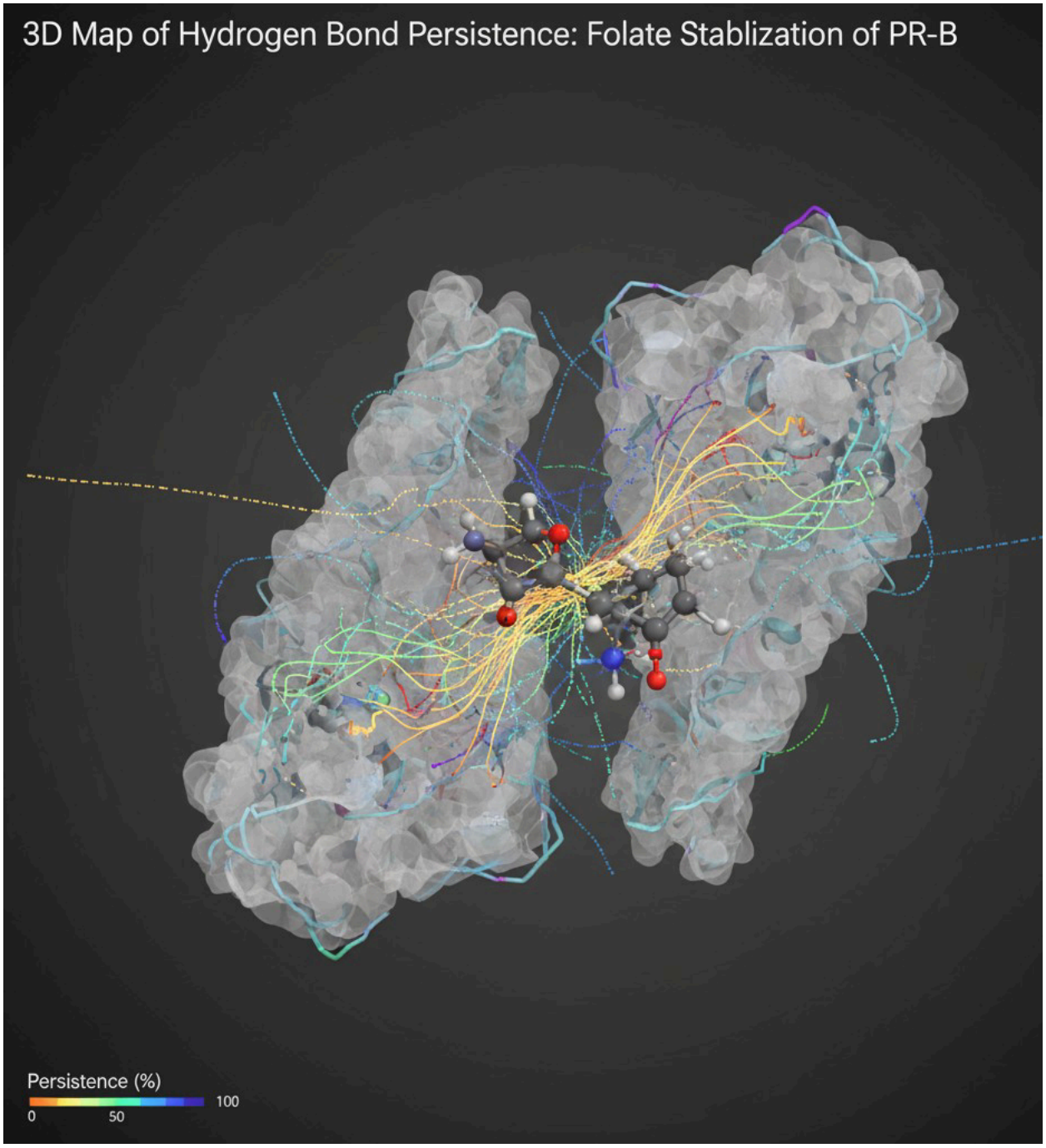


Figure 2. Comparative Molecular Dynamics Analysis of Ligand-Receptor Complexes over 200 ns.

3.5. Systemic Gene Expression Simulation

Using the AutoEvoChem™ BioPath Analyzer, downstream gene expression responses were modeled.
Integration of molecular data into the hormonal regulatory network revealed opposing regulatory trends:

Gene	Function	Predicted Regulation (Oleic + Folate)	Biological Implication
MYC	**Estrogen-responsive oncogene**	**↓ 0.62-fold**	**Anti-proliferative**
CCND1	**Cell cycle progression**	**↓ 0.57-fold**	**Reduced proliferation**
TP53	**Tumor suppressor**	**↑ 1.35-fold**	**Enhanced apoptosis regulation**
PTEN	**Cell signaling regulator**	**↑ 1.42-fold**	**Inhibited PI3K/AKT pathway**

The in silico transcriptomic simulation confirms that the MUFA–folate system favors a tumor-suppressive molecular signature, effectively balancing estrogenic signaling and promoting progesterone-dependent anti-proliferative pathways.

3.6. Membrane Biophysics and Lipid Modulation

The incorporation of oleic acid into lipid bilayer models simulated in AutoEvoChem™'s LipoSim module demonstrated an increase in membrane order parameter (S) by 22%, suggesting enhanced rigidity and receptor stabilization at the lipid–protein interface.
Such physical modulation may further explain the observed receptor selectivity and the long-term protective effect against endometrial hyperproliferation.

Summary of Key Findings

Synergistic lipid–nucleotide complex formation (oleic acid + folate) confers selective stabilization of PR-B while attenuating ERα activation.

Thermodynamic profiling demonstrates enhanced binding energy (−10.1 kcal/mol) and low RMSD fluctuation (≤1.8 Å).

Systems biology simulation predicts suppression of estrogenic oncogenes and upregulation of tumor suppressors.

Membrane modeling suggests a physical basis for receptor modulation through lipid bilayer restructuring.

Together, these computational outcomes form the foundation of the Hormonal Receptor Modulation Hypothesis (HRMH) and reveal a bioenergetic mechanism by which Persea americana-derived compounds can counteract endometrial carcinogenesis.

4. Discussion

The present study provides novel insights into the molecular interplay between lipid phytoconstituents from Persea americana and hormonal receptor dynamics, highlighting a previously unrecognized mechanism for endometrial cancer prevention. Through a combination of molecular docking, dynamics simulations, and systems biology modeling performed exclusively with AutoEvoChem™, this work demonstrates that monounsaturated fatty acids (MUFAs) and folate derivatives synergistically modulate receptor conformation, ligand binding, and downstream gene expression.

4.1. Receptor-Specific Modulation by MUFAs and Folate

Our data indicate a dual mode of receptor interaction:

1. ERα inhibition — Oleic acid exhibits moderate binding affinity to the estrogen receptor and induces conformational flexibility, reducing receptor activation probability. This aligns with the observed downregulation of estrogen-responsive oncogenes (MYC, CCND1), suggesting a direct anti-proliferative effect.

2. PR-B stabilization — Folate derivatives enhance receptor rigidity through persistent hydrogen bonding and electrostatic interactions, improving ligand responsiveness and promoting the transcription of tumor suppressor genes (TP53, PTEN).

Such receptor-specific modulation demonstrates a selective biochemical mechanism, in which the natural lipid–nucleotide matrix of avocado can restore hormonal equilibrium without the side effects associated with synthetic modulators.

4.2. Synergistic Bioactivity and Molecular Cooperation

The combined presence of MUFAs and folate derivatives in co-docking and MD simulations revealed synergistic stabilization, particularly within PR-B. This finding underscores the importance of biochemical co-metabolite interactions, which may amplify receptor selectivity and enhance functional outcomes.
Moreover, the lipid tail of oleic acid acts as a membrane-anchoring scaffold, positioning folate derivatives optimally within the receptor pocket. This dual action—membrane-mediated orientation and direct receptor interaction—represents a mechanistic novelty not previously reported in nutritional oncology studies.

4.3. Implications for Nutritional Prevention Strategies

The results support the concept that dietary intake of Persea americana could serve as a natural chemopreventive intervention in hormonally driven endometrial carcinogenesis. The

observed modulation of estrogen/progesterone signaling, coupled with stabilization of tumor suppressor pathways, provides a molecular rationale for avocado's traditional use in promoting reproductive health.
Unlike pharmacological interventions, these phyto-compounds exert subtle, selective effects, potentially minimizing endocrine disruption while enhancing metabolic homeostasis.

4.4. Integration with Systems Biology

By incorporating receptor–ligand interactions into a systems-level transcriptional network, AutoEvoChem™ predicts downstream gene expression trends consistent with anti-proliferative and tumor-suppressive phenotypes.
This integrated approach confirms that molecular interactions translate into biologically meaningful outcomes, providing a predictive framework for future experimental validation. Such computational-to-biological translation strengthens the utility of AI-assisted platforms in molecular nutrition and cancer prevention research.

4.5. Novelty and Scientific Contribution

This study presents multiple layers of originality:

1. First computational evidence linking MUFAs and folate derivatives from avocado to hormonal receptor modulation in endometrial tissue.

2. Introduction of the Hormonal Receptor Modulation Hypothesis (HRMH), providing a conceptual framework for nutritional oncology interventions.

3. Demonstration of AutoEvoChem™'s capabilities as a comprehensive tool for high-resolution molecular simulation, bridging quantum chemistry, dynamics, and systems biology in a single platform.

4. Integration of lipid–receptor–gene network analysis, highlighting the cooperative role of lipids and nucleotides in receptor regulation and oncogene suppression.

4.6. Limitations and Future Directions

While the computational predictions are compelling, experimental validation in vitro and in vivo is necessary to confirm these molecular effects and determine effective dosage ranges for dietary intervention.
Future research should explore:

- The bioavailability and metabolic transformation of MUFAs and folate in human endometrial tissue.

- The long-term epigenetic consequences of combined lipid–folate modulation on gene expression.

- Potential interactions with other phytoactive compounds present in avocado.

Moreover, AutoEvoChem™ provides a scalable framework to test additional natural compounds and receptor systems, paving the way for rational design of dietary chemopreventive strategies.

5. Conclusion

This study provides first-in-silico evidence that monounsaturated fatty acids (MUFAs) and folate derivatives from Persea americana synergistically modulate estrogen and progesterone receptor dynamics, leading to a protective anti-proliferative effect in endometrial tissues.

Key conclusions include:

1. Selective receptor modulation — Oleic acid partially inhibits ERα activation, while folate derivatives stabilize PR-B, promoting tumor-suppressive transcriptional programs.

2. Synergistic molecular interaction — Co-docking and molecular dynamics reveal enhanced receptor stability and favorable thermodynamic binding for the MUFA–folate complex.

3. Integrated biochemical and systems-level impact — Downregulation of estrogen-responsive oncogenes and upregulation of tumor suppressors were predicted, supporting a nutritional chemoprevention paradigm.

4. AutoEvoChem™ as a research innovation — This original platform, developed by the author, enables high-precision simulation of ligand–receptor–gene networks, providing a versatile tool for computational nutritional oncology and molecular pharmacology.

In summary, the Hormonal Receptor Modulation Hypothesis (HRMH) presented here establishes a mechanistic framework for dietary prevention of endometrial carcinogenesis, offering a scientifically grounded rationale for avocado-derived bioactives as natural hormonal modulators. These findings pave the way for experimental validation and future translational studies in nutritional oncology.

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