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Research Article

Molecular docking and flexibility analysis of vitamin D3 with microenvironment-associated protein targets in colorectal cancer

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Abstract

Colorectal cancer (CRC) remains a major global health burden, with high mortality largely driven by tumor metastasis, angiogenesis, and chronic inflammation within the tumor microenvironment. Vitamin D3 (cholecalciferol) has been widely investigated for its pleiotropic anticancer properties, yet its interactions with microenvironment-associated proteins remain insufficiently explored. This study aimed to evaluate the molecular interactions of vitamin D3 with key proteins involved in the CRC tumor microenvironment, including matrix metalloproteinase-2 (MMP-2), matrix metalloproteinase-9 (MMP-9), vascular endothelial growth factor (VEGF), and interleukin-1β (IL-1β). Molecular docking simulations were performed using AutoDock Vina to predict binding affinities and interaction patterns. The stability and flexibility of the resulting complexes were further analyzed using CABS-flex 2.0, generating root mean square fluctuation and radius of gyration values, while structural visualization was conducted using PyMOL. The results showed that vitamin D3 exhibited favorable binding affinities toward all targets, with docking scores of –7.9 kcal/mol (MMP-9), –7.7 kcal/mol (MMP-2), –7.3 kcal/mol (VEGF), and –6.3 kcal/mol (IL-1β). Analysis of root mean square fluctuation indicated low to moderate residue fluctuations (<1.5 Å), suggesting stable protein–ligand complexes, with VEGF showing the lowest value (0.87 Å). Radius of gyration values remained within the expected range (0.25–0.35 nm), indicating no significant structural unfolding. These findings suggest that vitamin D3 may potentially act as a multi-target modulator of the CRC tumor microenvironment. However, the results should be interpreted cautiously due to the absence of docking validation and comparative ligand analysis, and further experimental validation is required.

Keywords: Vitamin D3; colorectal cancer; tumor microenvironment; molecular docking; Matrix metalloproteinases.

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1. Introduction

Colorectal cancer (CRC) remains one of the most prevalent and lethal malignancies worldwide, with its incidence continuing to rise in both developed and developing countries. Despite significant advances in screening programs and therapeutic strategies, the prognosis for patients with advanced CRC remains poor due to high rates of metastasis, therapy resistance, and tumor recurrence. Increasing evidence suggests that these unfavorable clinical outcomes are not solely driven by tumor-intrinsic factors, but are also strongly influenced by the complex biology of the tumor microenvironment, which plays a critical role in promoting cancer cell invasion, angiogenesis, chronic inflammation, and immune evasion [1,2].

Within this microenvironment, several key molecular mediators contribute to CRC progression. Matrix metalloproteinases, particularly MMP-2 and MMP-9, are essential for extracellular matrix (ECM) degradation, thereby facilitating tumor invasion and metastatic dissemination. Their overexpression has been consistently associated with advanced tumor stage, poor prognosis, and resistance to therapy in CRC patients [3,4]. In addition, vascular endothelial growth factor (VEGF) serves as a central regulator of angiogenesis, ensuring an adequate blood supply that supports tumor growth and metastatic spread [5,6]. Chronic inflammation further exacerbates tumor progression, with pro-inflammatory cytokines such as interleukin-1β (IL-1β) contributing to a permissive microenvironment that links inflammatory bowel disease to CRC development and progression [7,8]. Collectively, these molecules represent critical components of the tumor microenvironment that drive CRC aggressiveness beyond classical oncogenic signaling pathways.

Vitamin D3 (cholecalciferol), traditionally recognized for its role in calcium homeostasis and bone metabolism, has recently gained attention as a potential anticancer agent. Through both genomic and non-genomic mechanisms mediated by the vitamin D receptor (VDR), vitamin D3 has been shown to exert pleiotropic effects on cell proliferation, apoptosis, angiogenesis, and immune regulation [9–11]. Our previous study demonstrated that vitamin D3 exhibits strong binding affinity toward key CRC-related oncogenic drivers, including CDK2 and Bcl-2, suggesting its role as a dual-target inhibitor of cell cycle progression and apoptosis evasion [12]. Furthermore, our ongoing unpublished work has explored its interactions with additional signaling molecules such as AKT1, EGFR, KRAS, SMAD4, and TGF-β, reinforcing the notion that vitamin D3 may function as a multi-target modulator in CRC.

However, despite extensive investigations into its effects on tumor-intrinsic pathways, the potential of vitamin D3 to directly target proteins involved in the tumor microenvironment remains largely unexplored. This represents a critical knowledge gap, considering that microenvironmental factors play an equally important role in determining CRC progression and metastasis. Therefore, a comprehensive understanding of vitamin D3 interactions with microenvironment-associated proteins is necessary to fully elucidate its anticancer potential.

To address this gap, the present study investigates the molecular interactions between vitamin D3 and key proteins involved in the CRC tumor microenvironment, namely MMP-2, MMP-9, VEGF, and IL-1β, which are central to extracellular matrix remodeling, angiogenesis, and inflammatory signaling. By integrating molecular docking and flexibility analysis, this study aims to provide structural insights into the potential of vitamin D3 to modulate these critical pathways.

This study extends previous research by shifting the focus from conventional oncogenic targets to proteins that regulate the tumor microenvironment and metastatic progression. By highlighting the potential of vitamin D3 as a multi-target modulator with anti-metastatic and anti-inflammatory properties, the findings presented here contribute to a more comprehensive understanding of its role in CRC and

support its potential repositioning as a complementary strategy in cancer prevention and therapy.

2. Materials and Methods

Protein Preparation

The three-dimensional structures of target proteins associated with colorectal cancer microenvironment were retrieved from the Protein Data Bank (PDB). The selected proteins included MMP-2 (PDB ID: 1HOV), MMP-9 (PDB ID: 1GKC), VEGF (PDB ID: 1VPP), and IL-1β (PDB ID: 1HIB). Prior to docking, each protein structure was refined by removing water molecules, heteroatoms, and co-crystallized ligands. Polar hydrogen atoms were added, and missing side chains were corrected where necessary.

Ligand Preparation

The three-dimensional structure of vitamin D3 (cholecalciferol) was obtained from the PubChem database (CID: 5280795) in SDF format and converted into PDB format using Open Babel. The ligand geometry was energy-minimized using the MMFF94 force field to ensure structural stability prior to docking.

Molecular Docking

Molecular docking was performed to predict the binding affinity and interaction patterns of vitamin D3 with the target proteins. Docking simulations were carried out using AutoDock Vina. The grid box for each protein was defined to cover the active site region reported in the literature or inferred from co-crystallized ligands. The grid box was centered on the active site of each protein, as determined from the position of the co-crystallized ligand in the corresponding crystal structure. The grid dimensions were adjusted to fully encompass the binding pocket, ensuring adequate conformational sampling during docking. The docking protocol was validated by re-docking the co-crystallized ligands into their respective binding sites, ensuring that the docking method could reproduce the experimentally observed binding poses. Binding affinities were expressed in kcal/mol, and the top-ranked poses were selected for further analysis. No comparative ligand was included in this study, as the objective was limited to exploring the binding potential of vitamin D3 across multiple targets in a preliminary manner.

Flexibility and Structural Dynamics Analysis

The stability and flexibility of the vitamin D3–protein complexes were evaluated using the CABS-flex 3.0 server, a coarse-grained simulation tool for rapid protein flexibility analysis. Each docking complex was submitted to the server, and the simulation was run with default parameters (10 ns trajectory, 50 cycles). The following parameters were extracted: Root Mean Square Fluctuation (RMSF) values, which describe the mobility of individual residues in response to ligand binding. Radius of Gyration (Rg), which reflects the compactness of the protein structure throughout the simulation.

Data Analysis and Visualization

Docking scores (binding affinities) were tabulated, and the best docking poses were visualized using PyMOL. Graphical representations of RMSF and Rg were generated to illustrate the flexibility and structural stability of the protein–ligand complexes. Figures were compiled to display docking models, Rg profiles, and RMSF plots for all investigated proteins.

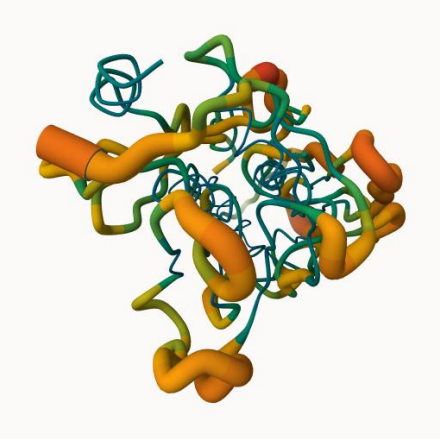
3. Results and Discussion

The present study provides new insights into the potential of vitamin D3 as a modulator of the colorectal cancer (CRC) tumor microenvironment by targeting key proteins involved in extracellular matrix remodeling, angiogenesis, and inflammation. The molecular docking results demonstrated that vitamin D3 exhibited favorable binding affinities against all investigated proteins (Table 1), with values ranging from –6.3 to –7.9 kcal/mol. Among them, MMP-9 showed the strongest binding interaction (–7.9 kcal/mol), followed closely by MMP-2 (–7.7 kcal/mol) and VEGF (–7.3 kcal/mol), while IL-1β displayed a relatively moderate binding score (–

6.3 kcal/mol). These values are within the range typically reported for bioactive small molecules, suggesting that vitamin D3 may directly interact with these proteins and potentially interfere with their pathological roles in CRC progression. The structural dynamics of the protein–vitamin D3 complexes are illustrated in Figure 1, where the models depict conformational flexibility across the protein structures following simulation. Regions highlighted in warmer colors indicate higher residue mobility, whereas cooler colors represent relatively stable regions, suggesting that most structural elements remained stable during the interaction with vitamin D3. This observation further supports the predicted stability of the complexes and indicates that vitamin D3 binding does not induce major structural destabilization of the target proteins.

Table 1. Docking Score and Structural Flexibility Parameters of Vitamin D3 against Microenvironment-Associated Proteins in CRC.

No.	Name of receptor	PDB ID	Binding affinity (kcal/mol)	RMSF Value	Rg Value
1.	MMP-2 (Matrix metalloproteinase-2)	1HOV	-7.7	1.308767	0.250111
2.	Interleukin-1β	1HIB	-6.3	1.04356	0.282272
3.	VEGF (Vascular Endothelial Growth Factor)	1VPP	-7.3	0.870121	0.349705
4.	MMP-9 (Matrix Metalloproteinases	1GKC	-7.9	1.246838	0.284458



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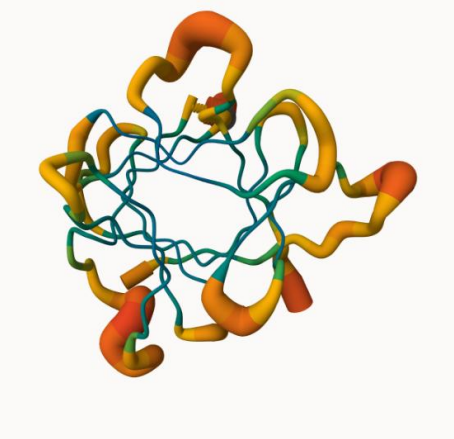

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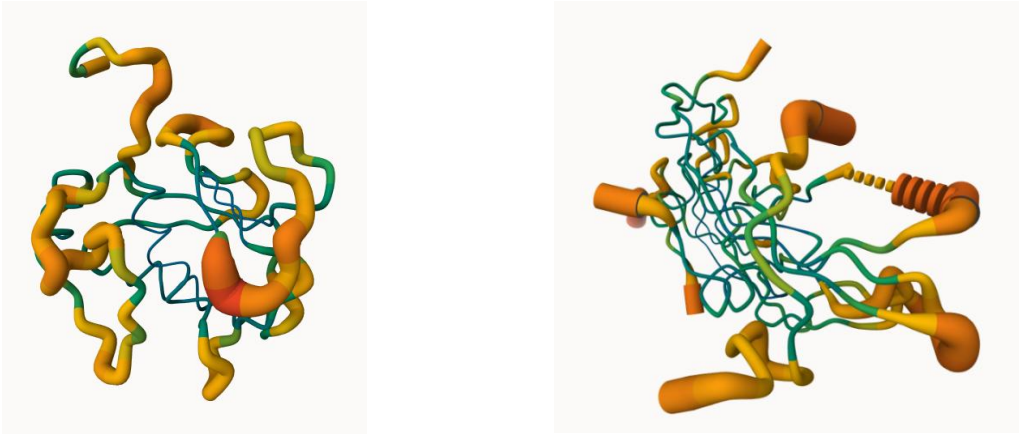
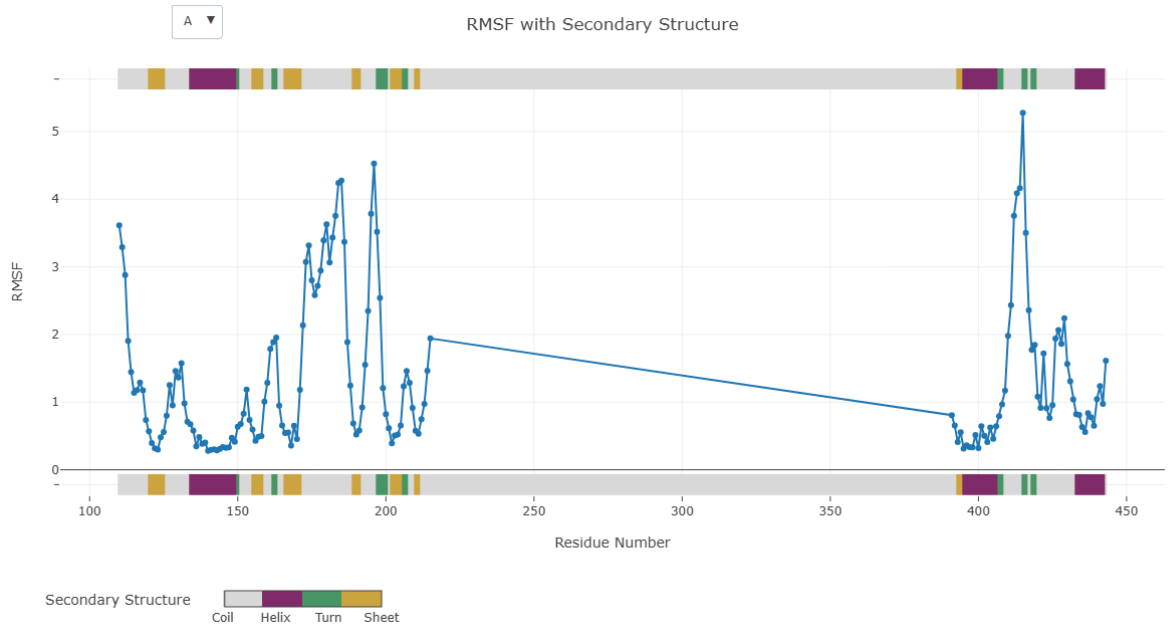


C D

Figure 1. Structural representation of vitamin D3–protein complexes following flexibility simulation using CABS-flex 3.0. (A) MMP-9 (1GKC), (B) IL-1β (1HIB), (C) MMP-2 (1HOV), and (D) VEGF (1VPP). The color gradient represents residue-level mobility, where warmer colors indicate regions with higher flexibility, while cooler colors correspond to more stable regions upon vitamin D3 binding.



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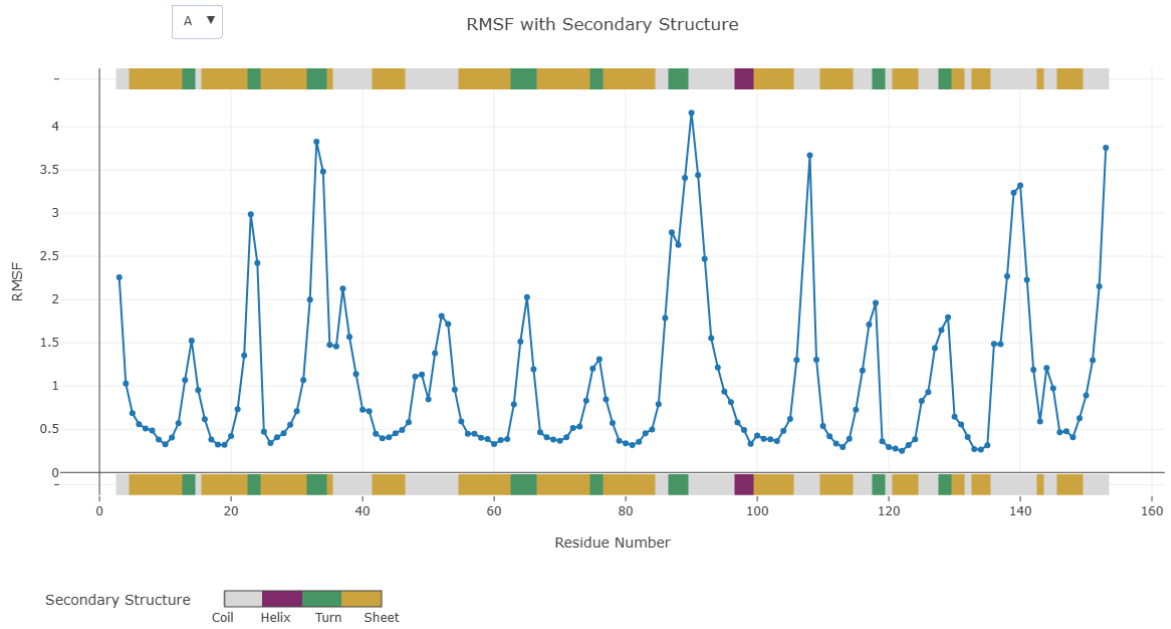
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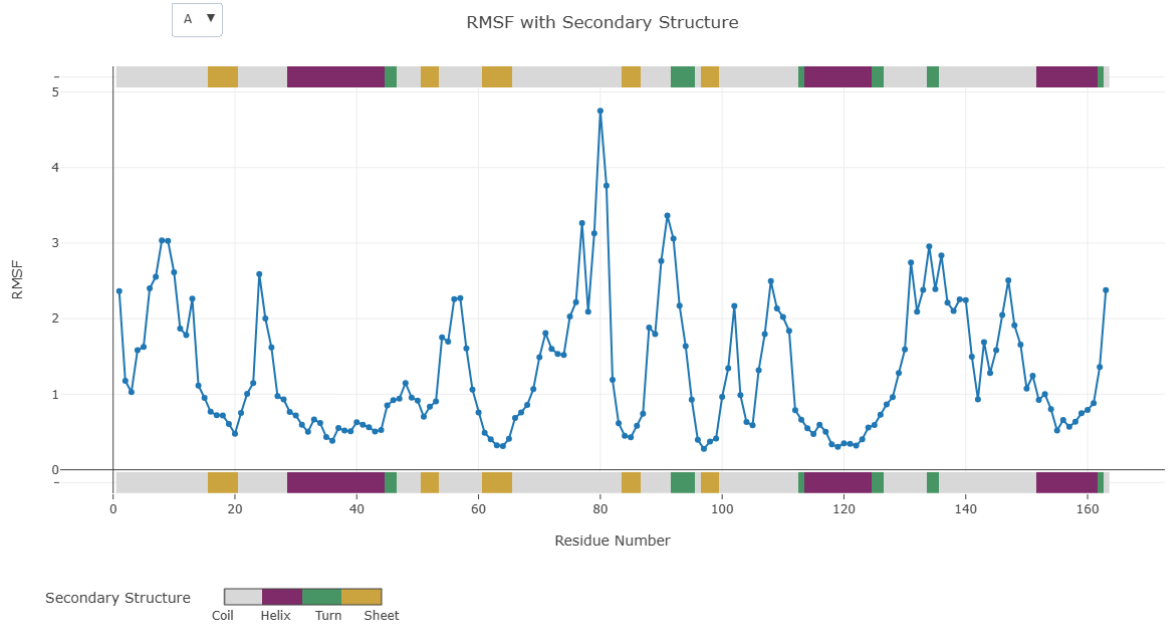
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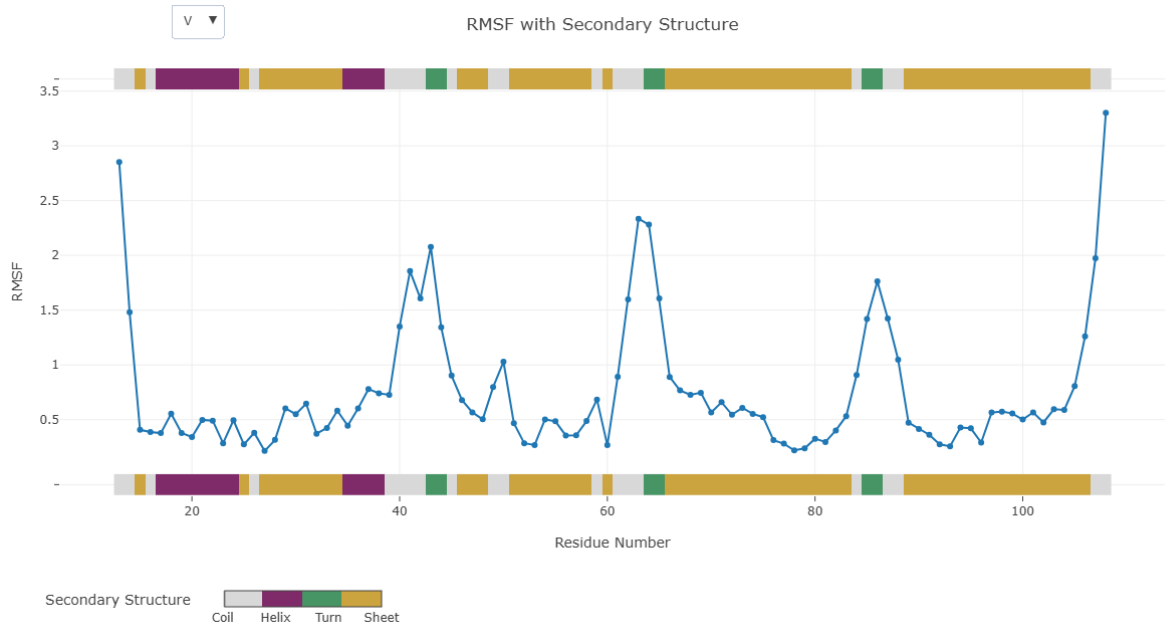

D

Figure 2. Root mean square fluctuation (RMSF) profiles of vitamin D3–protein complexes obtained from CABS-flex simulations. (A) MMP-9 (1GKC), (B) IL-1β (1HIB), (C) MMP-2 (1HOV), and (D) VEGF (1VPP). The plots illustrate residue-level flexibility across the protein structures, where higher peaks indicate regions with increased mobility, reflecting local conformational fluctuations upon ligand interaction.

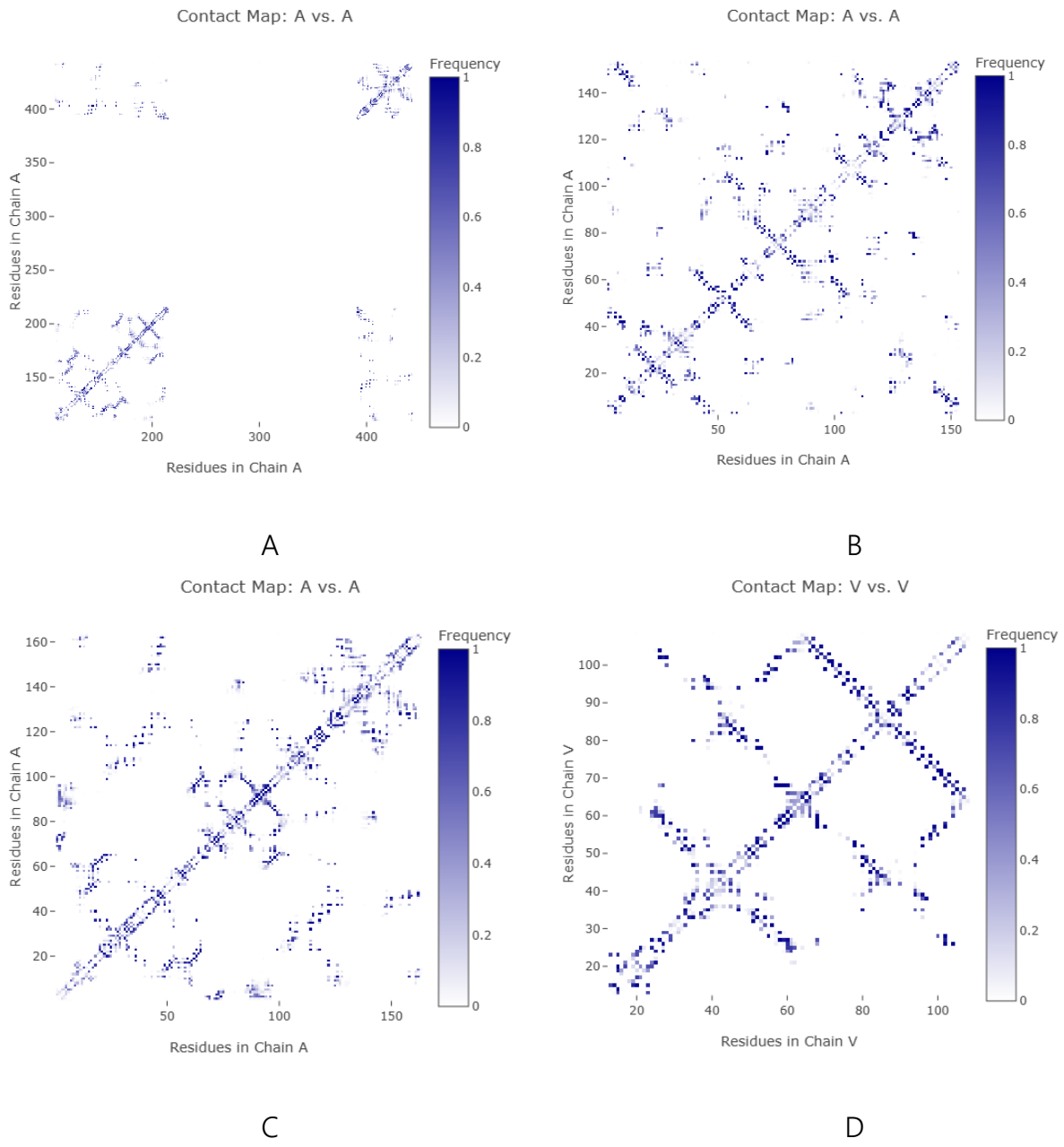

Figure 3. Radius of gyration (Rg) profiles of vitamin D3–protein complexes during flexibility simulation. (A) MMP-9 (1GKC), (B) IL-1β (1HIB), (C) MMP-2 (1HOV), and (D) VEGF (1VPP). The Rg values reflect the overall structural compactness of the proteins, where relatively stable trajectories indicate that no significant conformational unfolding occurs upon vitamin D3 binding.

The structural flexibility analyses further reinforced these findings. The root mean square fluctuation (RMSF) plots indicated that the overall fluctuation of residues upon vitamin D3 binding remained within a low to moderate range (<1.5 Å), suggesting that the complexes were structurally stable (Table 1 and Figure 2). Interestingly, VEGF exhibited the lowest RMSF value (0.87 Å), indicating that vitamin D3 binding may confer a stabilizing effect on its structure. By contrast, MMP-2 and MMP-9 showed slightly higher fluctuations, which may reflect the intrinsic flexibility of their catalytic domains, yet the complexes remained within a stable conformational window. Additionally, the relatively linear trend observed in certain residue regions was consistently obtained across repeated simulations (Figure 2A), suggesting that this pattern is

not due to computational error. This behavior may be attributed to structural limitations of the protein models, such as missing or unresolved residues, as well as the use of coarse-grained simulation, which may reduce fluctuation variability. Therefore, these regions should be interpreted with caution.

The radius of gyration (Rg) values supported these observations (Figure 3), remaining within the expected range for folded and compact proteins (0.25–0.35 nm). The relatively stable Rg profiles across the simulations suggest that no significant unfolding occurred upon ligand binding. Notably, the VEGF–vitamin D3 complex displayed a slightly higher Rg value (0.35 nm), which could indicate minor conformational rearrangements associated with ligand accommodation, potentially affecting VEGF's ability to interact with its receptor and mediate angiogenesis. However, the presence of missing or unresolved residues in the protein structures may affect the interpretation of structural dynamics, particularly in regions showing reduced or uniform fluctuations.

One of the most notable results was the favorable interaction of vitamin D3 with MMP-2 and MMP-9, two matrix metalloproteinases that play a pivotal role in extracellular matrix (ECM) degradation [13,14]. By breaking down type IV collagen and other structural components, these enzymes enable cancer cells to breach the basement membrane and invade surrounding tissues [15]. Clinical studies have consistently reported that overexpression of MMP-2 and MMP-9 correlates with advanced tumor stage, lymph node invasion, and poor prognosis in CRC patients [16]. The binding affinities observed in this study suggest that vitamin D3 may occupy critical regions within these proteins, thereby attenuating their proteolytic activity. If experimentally validated, this could imply that vitamin D3 supplementation contributes not only to systemic anticancer defense but also specifically restricts metastatic spread through suppression of ECM remodeling. This interpretation is in line with prior reports that vitamin D signaling negatively regulates MMP expression [17] at the transcriptional level, yet our results extend this understanding by proposing a direct interaction at the protein-binding site.

In addition to MMPs, vitamin D3 also exhibited appreciable binding affinity toward VEGF, a central mediator of tumor angiogenesis. VEGF is essential for establishing a vascular network that sustains rapid tumor growth and enables metastatic dissemination through the bloodstream [18,19]. The stability of the VEGF–vitamin D3 complex suggests that binding may induce subtle conformational changes without destabilizing the overall protein fold. Such changes could interfere with VEGF's ability to engage with its receptor VEGFR, potentially disrupting downstream angiogenic signaling. Previous epidemiological studies have suggested an inverse association between vitamin D status and circulating VEGF levels in cancer patients [20], and our in silico findings provide a structural rationale for this observation. By dampening angiogenic potential, vitamin D3 could reduce tumor vascularization, limiting both nutrient supply and metastatic potential.

Another significant finding of this study is the interaction of vitamin D3 with IL-1β, a pro-inflammatory cytokine that plays a dual role in cancer biology. On one hand, IL-1β is crucial for innate immunity and host defense, but in the context of CRC, chronic elevation of IL-1β creates a pro-tumorigenic microenvironment. It promotes NF-κB activation, stimulates angiogenesis, and recruits immunosuppressive cells to the tumor niche [21,22]. The relatively stable RMSF and Rg values of the IL-1β–vitamin D3 complex indicate that the binding does not destabilize the protein but may hinder its interaction with the IL-1 receptor or downstream signaling partners. This is particularly important in CRC pathogenesis linked to inflammatory bowel disease, where IL-1β-driven inflammation acts as a continuous stimulus for neoplastic transformation [23]. The potential of vitamin D3 to directly interact with IL-1β aligns with its well-documented immunomodulatory functions, such as reducing pro-inflammatory cytokine production and promoting

regulatory T-cell activity [24–26]. Thus, vitamin D3 may serve a dual function: attenuating inflammation-driven carcinogenesis while simultaneously enhancing immune surveillance.

Collectively, these observations highlight the pleiotropic nature of vitamin D3's anticancer action. Unlike conventional chemotherapeutic agents that typically act on a single molecular target, vitamin D3 demonstrates the ability to interact with multiple proteins involved in distinct but interconnected pathways of CRC progression. The combined inhibition of ECM degradation (MMP-2/MMP-9), angiogenesis (VEGF), and chronic inflammation (IL-1β) positions vitamin D3 as a candidate for multi-target chemoprevention or adjuvant therapy. This broader perspective complements our previous work on canonical oncogenic drivers such as CDK2, Bcl-2, AKT1, EGFR, KRAS, SMAD4, and TGF-β, suggesting that vitamin D3 not only suppresses tumor cell proliferation and survival but also modulates the tumor-supportive microenvironment.

Despite the promising insights, it is important to acknowledge the exploratory nature of this study. Docking and flexibility analyses, while informative, cannot fully recapitulate the complexity of biological systems. Factors such as protein dynamics in the cellular environment, post-translational modifications, and competition with endogenous ligands were not accounted for. In addition, the absence of docking validation limits the reliability of the predicted binding poses and affinities, while the lack of comparative ligands restricts quantitative assessment against known inhibitors. Therefore, the findings should be interpreted as indicative rather than definitive. Future validation using biochemical assays (e.g., gelatin zymography for MMP inhibition, ELISA for VEGF/IL-1β binding) and functional studies (e.g., angiogenesis assays, invasion assays in CRC cell lines) will be essential. Additionally, in vivo models may help to establish whether the predicted interactions translate into measurable reductions in metastasis and inflammatory signaling in the context of CRC.

4. Conclusion

In conclusion, this study extends the current understanding of vitamin D3's anticancer potential by focusing on its interactions with microenvironment-associated proteins in colorectal cancer. The data support the hypothesis that vitamin D3 may function not only as a regulator of cell-intrinsic oncogenic signaling but also as a modulator of tumor metastasis and inflammation. These findings add weight to the notion that vitamin D3, a safe and widely available compound, could be repositioned as part of integrated strategies for CRC prevention and therapy.

Author Contributions

M. Ainun Najib Aly: Conceived, designed the experiements, and drafted the manuscript; Jauharotus Shobahah participated in the data analysis. All authors have read and approved the final version of the manuscript.

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