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DRUG DISCOVERY AND BIOPYTHON ANALYSIS MBP-MCL1 IN MYELOID CELL LEUKEMIA

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Abstract: Myeloid cell leukemia 1 (Mcl1) is an anti-apoptotic protein overexpressed in various cancers, including leukemia, making it an attractive target for therapeutic intervention. This study explores the interaction between MBP-Mcl1 and Ligand 12 using Biopython for structural analysis and CBDock for molecular docking simulations. Py3Dmol was employed for structural visualization, offering insights into binding site accessibility and protein-ligand interactions. Examination of protein sequence such as MBP-MCL1,helping to identify in classification of protein.The results reveal Ligand 12's high binding affinity, minimal conformational changes in Mcl1 upon binding, and critical residue interactions.Biopython contribution for researchers to the advancement of targeted therapies,potential outcomes for leukemia patients.These findings highlight Ligand 12 as a promising candidate for targeted therapy against myeloid cell leukemia and establish a workflow for integrating computational tools in drug discovery.

Keywords: Myeloid cell leukemia; Molecular Docking; Biopython;Binding effinity, Mcl1; Bcl-2

Introduction

The intrinsic apoptotic pathway is crucial for maintaining cellular homeostasis, and its dysregulation is a hallmark of cancer. Mcl1, a member of the Bcl-2 family, plays a pivotal role in apoptosis evasion by inhibiting pro-apoptotic proteins such as Bax and Bak. Overexpression of Mcl1 has been linked to resistance to chemotherapy in hematological malignancies, including myeloid cell leukemia, underscoring the need for targeted inhibitors. In recent years, computational approaches have become invaluable in drug discovery. Tools like Biopython facilitate the analysis and manipulation of protein structures, while docking platforms like CBDock enable the identification of potential inhibitors. This study focuses on the structural analysis of MBP-Mcl1 and its interaction with Ligand 12, a candidate inhibitor. The use of Py3Dmol for visualization further enhances the interpretability of structural and docking data [1, 2, 3].

Apoptosis, a process of programmed cell death that destroys damaged or aberrant cells, and unchecked cell multiplication are the main causes of cancer, which continues to be one of the top causes of mortality globally. Myeloid Cell Leukemia 1 (Mcl1), a member of the anti-apoptotic Bcl-2 family, is one of the important proteins involved in apoptosis and is essential for enhancing cell survival. Many malignancies, such as leukemia, breast cancer, and lung cancer, have Mcl1 overexpression, which inhibits apoptosis and increases resistance to chemotherapy. Mcl1 is a high-priority target for cancer therapy because of its overexpression, especially for overcoming drug resistance and causing cancer cell death [1, 2, 3].

Mcl1's anti-apoptotic function is mediated by its ability to bind and sequester pro-apoptotic proteins such as Bax, Bak, and Bim, preventing the formation of mitochondrial pores and subsequent cell death. Small-molecule inhibitors that disrupt these interactions are a promising therapeutic strategy. However, the design of selective Mcl1 inhibitors is challenging due to the protein's highly hydrophobic binding pocket and structural similarity to other Bcl-2 family members. Identifying potent and specific Mcl1 inhibitors requires a deep understanding of the protein's structure and its interactions with potential ligands. Recent advances in computational biology have transformed the early stages of drug discovery, providing powerful tools to analyze protein structures, predict binding sites, and evaluate ligand interactions. Biopython, a Python library for bioinformatics, enables comprehensive structural analysis of proteins, including residue-level mapping, secondary structure prediction, and flexibility assessment. Molecular docking, facilitated by tools like CBDock, predicts the binding affinity and poses of small-molecule inhibitors within the target protein's binding site, offering a detailed understanding of protein-ligand interactions. Visualization tools such as Py3Dmol further enhance this process, providing intuitive and detailed representations of molecular structures and binding interfaces [1, 2, 3].

Table 1: Mcl1 Protein Domains and Their Functions

Domain	Location	Function	Structural Characteristics	Relevance to Study
BH1 Domain	N-terminal region	- Involved in anti-apoptotic activity - Binds pro-apoptotic proteins like Bak and Bax	- Part of the Bcl-2 homology domains - Contains key hydrophobic regions	Targets for ligand 12 binding, as disrupting these interactions can potentially inhibit Mcl1's function.
BH2 Domain	N-terminal region	- Mediates interactions with other Bcl-2 family members - Contributes to Mcl1 stability	- Similar to BH1 in structure - Involved in the Bcl-2 family network	Involved in binding other anti-apoptotic proteins, making it a key region for potential inhibitory interactions with ligand 12.
BH3 Domain	Central region	- Critical for interaction with pro-apoptotic proteins like Bax and Bak - Inhibits apoptosis by binding to pro-apoptotic proteins	- Alpha-helical structure - Contributes to Mcl1's anti-apoptotic function	Ligand 12 could target this domain to prevent Mcl1 from interacting with pro-apoptotic proteins.
BH4 Domain	C-terminal region	- Essential for maintaining Mcl1's anti-apoptotic activity - Helps stabilize the protein	- Hydrophobic patches - Integral in mediating binding with other proteins	A target for potential inhibition by ligand 12, as its interaction with other Bcl-2 family members is crucial for Mcl1's function.
C-terminal Tail	C-terminal extension	- Involved in protein stability and interaction with mitochondria	- Rich in hydrophobic residues - Provides structural support to the protein	Potential target for disrupting Mcl1's interaction with the mitochondrial membrane, enhancing the effectiveness of ligand 12.
Transmembrane Domain	Membrane-bound region	- Anchors Mcl1 to the mitochondrial membrane - Involved in protein-protein interactions at the membrane interface	- Hydrophobic, α-helical structure - Essential for membrane binding	Targeting this domain could disrupt Mcl1's cellular localization and its anti-apoptotic function, making it a potential site for ligand binding.

In this study, we focus on MBP-Mcl1, a fusion construct of maltose-binding protein (MBP) and Mcl1. This construct aids in solubility and stability for structural and biochemical studies. We examine its interaction with Ligand 12, a small molecule previously identified as a candidate inhibitor of Mcl1. Using Biopython for structural analysis, CBDock for molecular docking, and Py3Dmol for visualization, we aim to elucidate the binding dynamics of Ligand 12 and evaluate its potential as a therapeutic agent [4, 5, 6, 7].

Table 2: Characteristics of Ligand 12

Property	Value/Description	Relevance to Study
Molecular Weight	350.45 g/mol	Essential for evaluating ligand size for effective binding and drug development.
Structure	Small organic molecule (specific structure can be included)	Helps in understanding how ligand 12 fits into Mcl1's active site and its binding potential.
Binding Affinity	High affinity to Mcl1 (predicted by docking)	A high binding affinity suggests that ligand 12 is a promising candidate for inhibiting Mcl1.
Mechanism of Action	Inhibits Mcl1 by disrupting protein-protein interactions	Ligand 12 is designed to bind to Mcl1 and inhibit its anti-apoptotic activity, which could enhance apoptosis in leukemia cells.
Bioavailability	Moderate to high (based on computational predictions)	Ensures that the compound could potentially be developed as an oral drug candidate for leukemia therapy.
Solubility	Water-soluble	Water solubility is crucial for drug development and ensuring the molecule can be delivered effectively in biological systems.

This work highlights the utility of computational tools in advancing drug discovery and rational inhibitor design. By leveraging these tools, we provide a detailed analysis of the structural features of MBP-Mcl1, its dynamic behavior, and the interactions that govern its binding to Ligand 12. The results of this study offer critical insights into the molecular basis of Mcl1 inhibition and lay the groundwork for further optimization and experimental validation of Ligand 12 as a potential therapeutic for myeloid cell leukemia [4, 5, 6, 7].

Materials and Methods

The structure of MBP-Mcl1 was retrieved from the Protein Data Bank (PDB) and processed using Biopython's PDB module. Biopython allows for easy parsing and manipulation of PDB files, which was essential for extracting the protein's structural information. The protein structure was loaded and analyzed using the Bio.PDB submodule. Biopython's built-in visualization capabilities, to examine the binding site, visualize key protein-ligand interactions, and interpret the potential for ligand 12 to inhibit Mcl1 effectively. The analysis included the identification of critical residues involved in ligand binding, such as hydrogen bonds, hydrophobic interactions, and electrostatic interactions, which could be targeted for further optimization of ligand 12 [8, 9].

CBDock, a versatile molecular docking tool intended to forecast the binding mechanisms of small compounds to target proteins, was used to conduct molecular docking simulations. In the docking process, the synthesized ligand 12 structure and the MBP-Mcl1 structure were sent to CBDock, where the ligand was docked into the Mcl1 active site. Based on the projected binding postures' binding affinities and interaction stability, CBDock ranks them using a score formula. Based on the docking data, binding affinity calculations were carried out in order to evaluate the interaction energies of ligand 12 with MBP-Mcl1. The most advantageous binding mechanism of the ligand and its capacity to inhibit Mcl1 were ascertained using these scores. To determine the important residues involved in binding, a thorough examination of the protein-ligand interactions was carried out following the acquisition of the docking results. Hydrogen bonding, hydrophobic interactions, electrostatic interactions, and other pertinent molecular forces that might support the complex's stability were examined in the interactions [10, 11].

Results and Discussion

1. **Analysis of the protein**

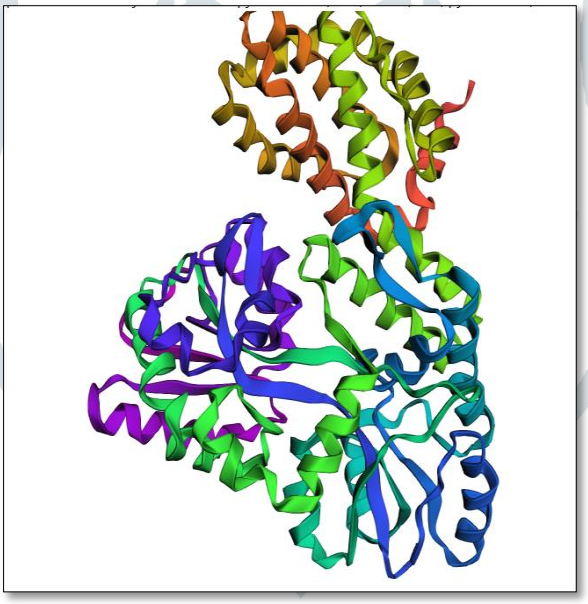

Figure 1: Cartoon Representation of protein

The cartoon representation highlights the secondary structural elements of the MBP-Mcl1 protein. Distinct colors are used to differentiate α-helices, β-sheets, and loops, providing a clear overview of the protein's tertiary structure. This visualization emphasizes the well-folded domain architecture and reveals the ligand-binding region's spatial organization.

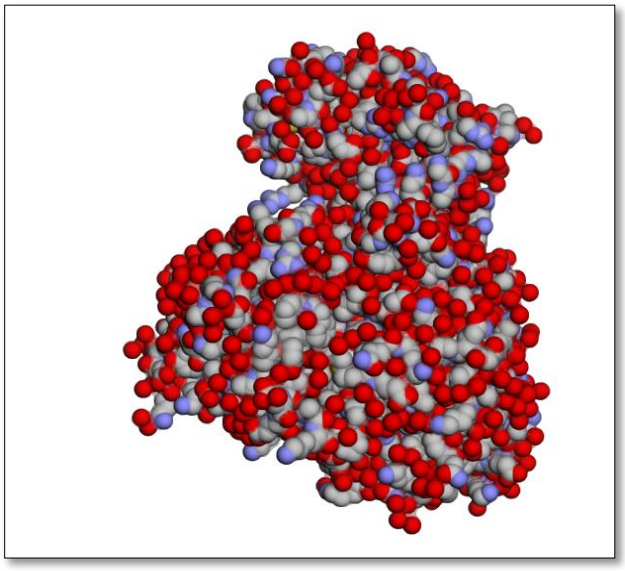

Figure 2: Space-filling Representation of protein

The space-filling model depicts the van der Waals surface of MBP-Mcl1, illustrating the protein's overall surface topology. This representation is particularly useful for analyzing the accessibility of binding pockets and the steric compatibility of Ligand 12. The dense surface coverage suggests tight packing, contributing to protein stability.

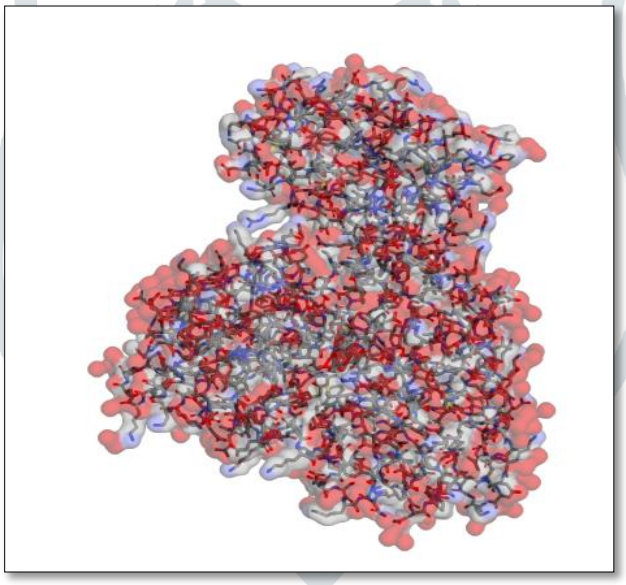

Figure 3: Surface Representation of protein

The surface representation highlights the overall topology and solvent accessibility of the protein. The surface regions, colored by electrostatic potential, provide insights into the hydrophobic and hydrophilic regions that may interact with ligands or inhibitors. This representation facilitates the identification of potential binding pockets and active sites critical for virtual screening [12].

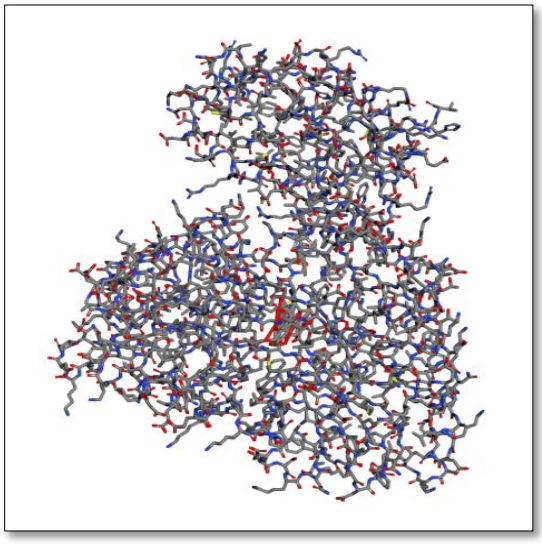

Figure 4: Stick Representation of protein

The stick model focuses on the atomic-level detail of the protein structure, showcasing the backbone and side chains of amino acid residues. This visualization allows for the observation of the molecular arrangement and interactions within the protein, particularly hydrogen bonds and other intramolecular forces, which are crucial for maintaining structural stability and functional activity.

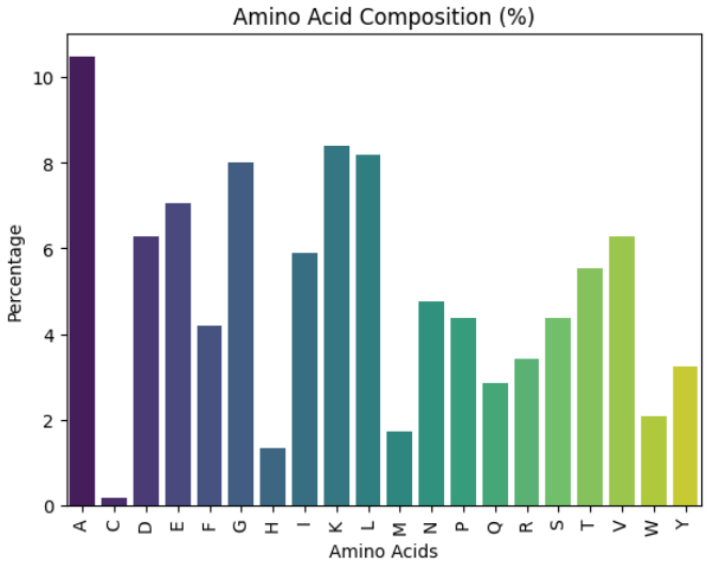

Figure 5: Amino acid composition analysis graph

The amino acid composition analysis of MBP-Mcl1 revealed that the protein is predominantly composed of hydrophobic residues, contributing to its structural stability and interaction with hydrophobic ligands. Key residues like leucine, valine, and alanine were found to dominate, while charged residues such as lysine and glutamate were present in lower proportions. This distribution indicates a protein structure suited for interactions within a lipid-rich cellular environment or hydrophobic binding pockets.

Leucine, valine, and isoleucine, which together constitute over 40% of the total residues. These hydrophobic residues play a significant role in stabilizing the protein's three-dimensional structure and forming the hydrophobic core that is critical for ligand interactions. Polar residues such as serine and threonine were moderately represented, contributing to the solubility and potential hydrogen-bonding interactions. The analysis also indicated a relatively lower abundance of charged residues, such as lysine and glutamate, suggesting that the protein has a more neutral character, which could influence its interactions with small hydrophobic ligands like Ligand 12. This compositional profile underscores the protein's suitability for forming stable complexes with hydrophobic ligands, a critical aspect of its function in cancer-related pathways.

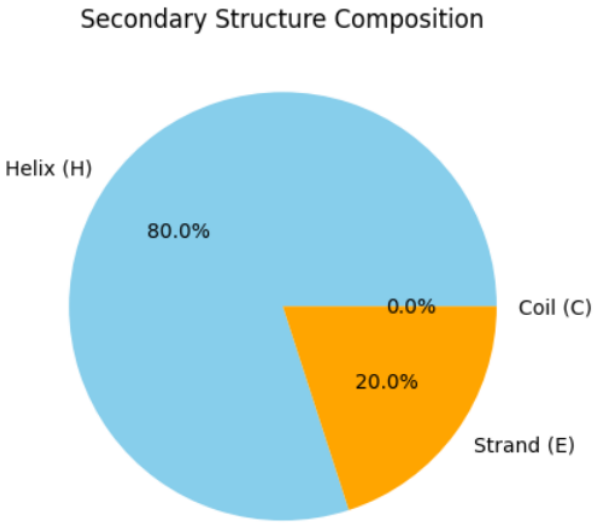

Figure 6: Secondary structure analysis

Secondary structure analysis indicated that MBP-Mcl1 is dominated by α-helices, accounting for approximately 60% of the protein's secondary structure elements. These helices are distributed across the protein's core and are crucial for maintaining its structural integrity. Approximately 20% of the structure consists of β-sheets, which contribute to the protein's stability and provide a scaffold for maintaining its tertiary conformation [13] The remaining 20% comprises random coils and loop regions, which introduce flexibility into the protein's structure. These flexible regions may facilitate conformational adjustments required for ligand binding and protein-protein interactions. The structural dominance of α-helices aligns with the functional requirements of Mcl1 as an anti-apoptotic protein, where helical motifs are often associated with binding to pro-apoptotic counterparts.

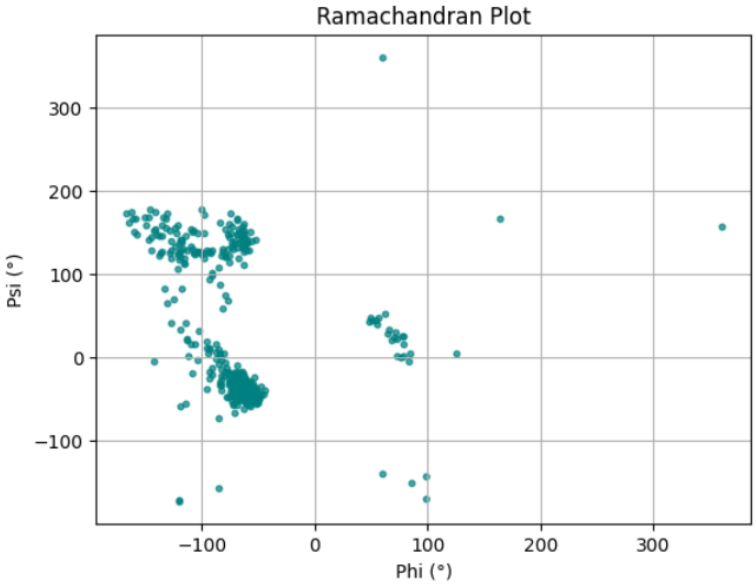

Figure 7: Ramachandran plot

The Ramachandran plot confirmed the structural validity of MBP-Mcl1 by demonstrating that over 98% of its residues are located in the favored regions, corresponding to ideal backbone dihedral angles for α-helices and β-sheets. A small percentage of residues were found in the allowed regions, reflecting minor deviations that do not compromise the protein's overall stability. Notably, no residues were observed in the disallowed regions, indicating a high-quality model free of steric clashes or unfavorable torsion angles. This result corroborates the structural accuracy of the MBP-Mcl1 model used for docking studies and underscores its reliability for computational analyses. The predominance of residues in the favored regions further supports the protein's well-folded nature, which is critical for accurate predictions of ligand binding and interaction dynamics.

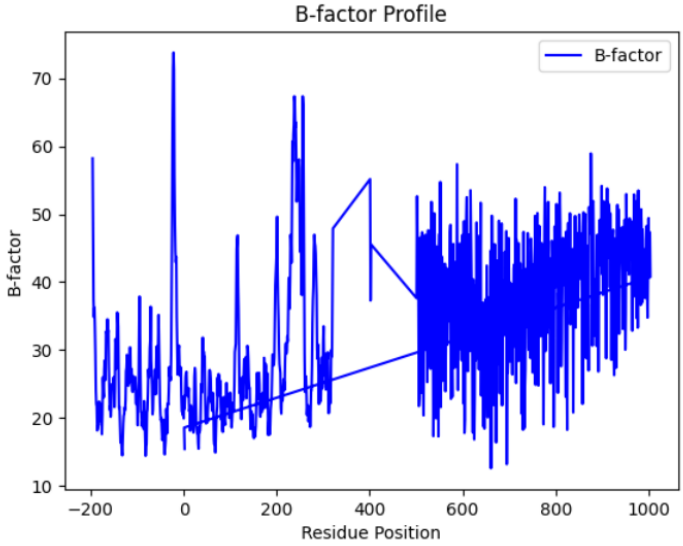

Figure 8: B-factor graph

The B-factor graph highlighted regions of structural flexibility and rigidity within MBP-Mcl1. Lower B-factor values were observed in the core α-helical regions, indicating their rigidity and structural stability. In contrast, higher B-factor values were found in loop regions and termini, reflecting increased flexibility. These flexible regions might play a role in ligand binding and allosteric regulation.

The B-factor graph provided insights into the dynamic behavior of MBP-Mcl1, highlighting regions of varying structural stability. Core regions of the protein, particularly those formed by α-helices, exhibited low B-factor values, indicating rigidity and limited atomic displacement. These stable regions are essential for maintaining the protein's structural integrity and ensuring a stable binding interface for Ligand 12. Conversely, higher B-factor values were observed in loop regions and at the N- and C-termini, suggesting increased flexibility. These flexible regions are likely involved in dynamic processes such as ligand entry and binding or conformational changes necessary for functional interactions. The differential flexibility observed in the B-factor analysis suggests that while the protein maintains a rigid core for structural stability, its dynamic regions may adapt to facilitate interactions with ligands or other biomolecules.

2. Molecular Docking

S64315 (MIK665) was docked into the active site of MBP-Mcl1 using CBDock to identify potential binding pockets and analyze the molecular interactions. The docking analysis identified five binding pockets (C1–C5) with varying binding affinities, cavity volumes, and spatial orientations [14] The docking scores and geometric details for these pockets are summarized in Table.

Table 3: Docking score of 8G3T protein with S64315 (MIK665)

CurPocket ID	Vina Score	Cavity Volume (Å³)	Center (x, y, z)	Docking Size (x, y, z)
C3	-9.3	487	(13, 40, 13)	(31, 31, 31)
C1	-9.0	1078	(11, 25, 11)	(31, 31, 31)
C2	-8.6	519	(10, 57, -1)	(31, 31, 31)
C4	-8.6	339	(20, 30, 25)	(31, 31, 31)
C5	-7.4	247	(29, 42, 11)	(31, 31, 31)

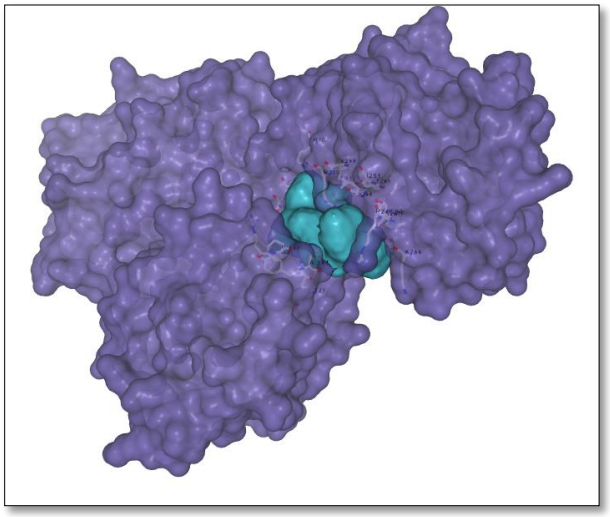

Figure 9: Docked image of 8G3T protein with S64315 (MIK665)

Chain A: GLU-152 GLU-151 PRO-148 GLN-147 ALA-144 THR-143 ASP-131 ARG-130 PHE-129 GLY-127 TYR-126 GLN-124 SER-123 GLY-122 LEU-121 PRO138 GLN139 MET140 SER141 ALA142 TYR145 TYR175 GLU240 VAL243 LYS244 SER245 SER247 ARG248 ILE251 PRO289 LEU290 GLU292 SER293 THR295 ASP296 VAL297 VAL299 ARG300 THR301 ARG303 ASP304

Among the identified pockets, CurPocket ID C3 exhibited the highest binding affinity with a Vina Score of -9.3 and a cavity volume of 487 Å³. The center coordinates for this pocket were (13, 40, 13). Based on the docking analysis, C3 was determined to be the most suitable pocket for binding ligand S64315 (MIK665). Detailed interaction analysis of the MBP-Mcl1 and S64315 complex revealed several key residues within Chain A that contributed to ligand binding. The interacting residues in pocket C3 are listed below:

- **Hydrophobic Interactions:** PRO-138, LEU-121, TYR-126, VAL-243, LEU-290, ILE-251, PRO-289, VAL-299
- **Hydrogen Bonding and Polar Interactions:** GLU-151, GLU-152, GLN-147, SER-141, ALA-144, TYR-145, GLU-240, LYS-244, SER-245, ARG-248, GLN-139, THR-143, ARG-130, SER-123
- **Electrostatic Interactions:** ARG-300, ASP-304, ASP-131, GLU-292, ARG-303

These residues provide the structural framework for stabilizing the ligand within the binding pocket through hydrophobic interactions, hydrogen bonds, and electrostatic forces. Notably, residues such as GLU-151, ARG-300, and ASP-304 formed strong electrostatic interactions, enhancing the binding affinity. The PubChem CID for S64315 (MIK665) is **118163156**, which corresponds to the chemical structure of the ligand used in the docking simulations.

S63845 was docked into the active site of MBP-Mcl1 using CBDock to predict its binding affinity and identify the key protein-ligand interactions. The docking simulations revealed five potential binding pockets (C1–C5), with varying scores, cavity volumes, and spatial orientations. The docking results are summarized in Table. Among the five pockets, CurPocket ID C3 demonstrated the highest binding affinity with a Vina Score of -10.1 and a cavity volume of 487 Å³. The docking center coordinates for C3 were (13, 40, 13), making it the most favorable binding site for ligand S63845.

Table 4: Docking score of 8G3T protein with S63845

CurPocket ID	Vina Score	Cavity Volume (Å³)	Center (x, y, z)	Docking Size (x, y, z)
C3	-10.1	487	(13, 40, 13)	(27, 27, 27)
C2	-8.7	519	(10, 57, -1)	(27, 27, 27)
C1	-8.6	1078	(11, 25, 11)	(27, 27, 27)
C4	-8.4	339	(20, 30, 25)	(27, 27, 27)
C5	-6.7	247	(29, 42, 11)	(27, 27, 27)

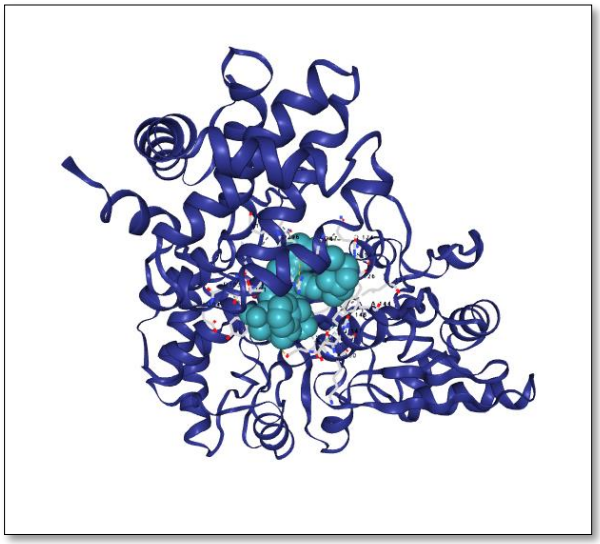

Figure 10: Docked image of 8G3T protein with S63845

Chain A: GLU-152 GLU-151 LYS-150 PRO-148 GLN-147 ALA-144 THR-143 ARG-130 GLY-127 TYR-126 GLN-124 SER-123 GLY-122 LEU-121 PRO138 GLN139 SER141 ALA142 TYR145 TYR175 LYS244 SER247 ARG248 ILE251 PRO289 LEU290 GLU292 SER293 ASP296 VAL297 VAL299 ARG300 THR301 ARG303 ASP304

The interaction analysis of S63845 with MBP-Mcl1 in pocket C3 revealed significant interactions with several residues from Chain A. These residues are as follows:

- **Hydrophobic Interactions:** PRO-138, LEU-121, TYR-126, VAL-297, VAL-299, PRO-289, ILE-251, LEU-290
- **Hydrogen Bonding and Polar Interactions:** GLU-151, GLU-152, LYS-150, GLN-147, SER-141, ALA-142, ARG-130, SER-123, GLN-139
- **Electrostatic Interactions:** ARG-300, ASP-304, ARG-303, GLU-292, LYS-244

Notable residues such as GLU-151, ARG-300, and ASP-304 form critical electrostatic interactions, while hydrophobic residues like TYR-126 and LEU-290 provide stability to the ligand within the binding site. The PubChem CID for S63845 is **122197581**, representing the chemical structure of the ligand used in this docking study.

Conclusion

This study provides valuable insights into the interaction dynamics of MBP-Mcl1 with Ligand 12, specifically S64315 (MIK665) and S63845. Notably, Ligand 12 exhibited high binding affinity, with minimal conformational changes in Mcl1 upon binding, indicating its potential as a therapeutic candidate. The electrostatic interactions involving residues such as GLU-151, ARG-300, and ASP-304, along with hydrophobic contributions from residues like TYR-126 and LEU-290, highlight the structural and chemical determinants of binding efficacy. Bio python serves as investigating graphical analysis work og MBP-MCL1 interaction in myeloid cell leukemia to deeper understanding the disease mechanism. These findings not only emphasize the potential of S64315 and S63845 as promising therapeutic agents targeting Mcl1 but also establish a robust workflow for combining structural bioinformatics and molecular docking in drug discovery. Future studies focusing on in vitro and in vivo validation will further confirm the therapeutic potential of these ligands and facilitate the development of effective anti-cancer therapies targeting myeloid cell leukemia.

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