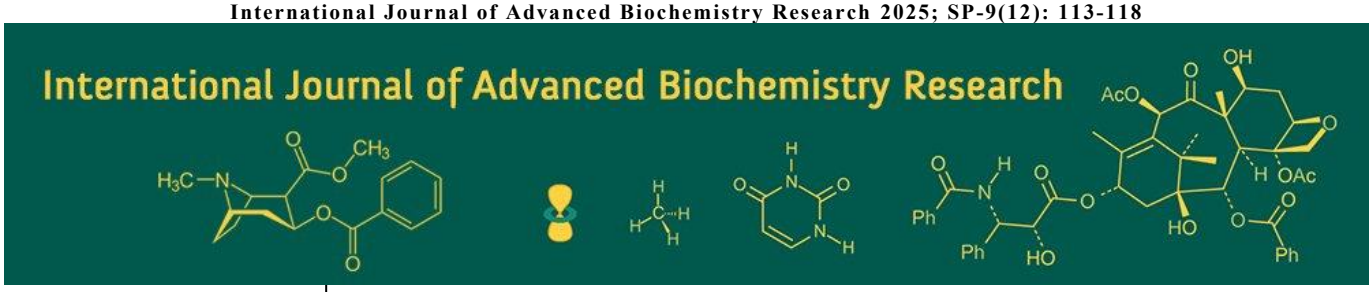

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Computational characterization of the tomato SUMO1-ToLCV AC2 complex illuminates SUMO-mediated viral manipulation of host immunity

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Abstract
Tomato Leaf Curl Virus (ToLCV) employs the multifunctional AC2 protein to manipulate host defense pathways, yet its potential interaction with the SUMOylation system remains unexplored. In this study, we investigated the structural basis and dynamic stability of the tomato SUMO1-AC2 complex using homology modeling, molecular docking, and long-timescale molecular dynamics simulations. Docking indicated favorable binding between SUMO1 and AC2, and MD simulations demonstrated that the complex remains stable, compact, and well-structured throughout 120 ns. RMSD, radius of gyration, surface area, and hydrogen-bond analyses confirmed the absence of large-scale unfolding or dissociation, while RMSF revealed flexibility mainly in loop regions of AC2. Secondary-structure profiles indicated retention of native folding patterns for both proteins. These findings suggest that AC2 can stably associate with SUMO1, potentially enabling ToLCV to interfere with SUMO-dependent immune regulation in tomato. The SUMO1-AC2 interface represents a promising target for engineering resistance against begomoviruses.

Keywords: SUMOylation system, tomato SUMO1 (or SUMO1 protein), ToLCV AC2 protein

Introduction
Plant viruses continue to impose major constraints on crop productivity worldwide, and among them, begomoviruses (family *Geminiviridae*) represent one of the most destructive groups infecting tomato (*Solanum lycopersicum*). Tomato Leaf Curl Virus (ToLCV) and closely related species such as Tomato Yellow Leaf Curl Virus (TYLCV) cause severe leaf curling, yellowing, reduced photosynthesis, stunting, and significant yield losses across tropical and subtropical regions (Navas-Castillo *et al*., 2011) [7]. These viruses encode a small number of multifunctional proteins that manipulate plant transcription, hormone signaling, epigenetic regulation, and antiviral defenses to establish a systemic infection (Hanley-Bowdoin *et al*., 2013; Guerrero *et al*., 2020) [4, 3].

Plants employ multiple layers of immunity against pathogens, many of which are modulated by post-translational modifications (PTMs). Among these, SUMOylation- the covalent attachment of Small Ubiquitin-Like Modifier (SUMO) proteins to target substrates-has emerged as a central regulator of transcription, chromatin remodeling, hormone signaling, protein stability, and stress responses (Miura & Hasegawa, 2010; Augustine & Vierstra, 2018) [6, 1]. The SUMOylation pathway involves an E1 activating enzyme (SAE1/SAE2), the E2 conjugating enzyme SCE1, multiple E3 ligases such as SIZ1 and MMS21, and SUMO proteases (ULPs) that remove SUMO from substrates. Many defense-associated proteins, including transcription factors, chromatin regulators, and the central immunity regulator NPR1, undergo SUMOylation during the activation of salicylic acid (SA)-mediated defense pathways (Saleh *et al*., 2015) [8]. These findings highlight the role of SUMOylation as an important layer of plant antiviral immunity. Recent evidence demonstrates that plant DNA viruses, including geminiviruses, directly manipulate the SUMOylation system to enhance infection. A landmark study showed that the Rep protein of a geminivirus interacts with components of the host SUMOylation cascade and suppresses SUMO conjugation, thereby weakening antiviral defenses (Arroyo-Mateos *et al*., 2018) [2]. Additional work has revealed that SUMO and SUMO-related proteins are dynamically regulated during viral infection and can influence host susceptibility or resistance (Jongkam *et al*., 2022) [5].

Although these studies were not performed in tomato, they establish a strong conceptual foundation indicating that geminiviruses exploit the SUMO pathway as part of their infection strategy.

Despite extensive research on SUMOylation in *Arabidopsis* and other model plants, very little is known about how SUMO modification influences ToLCV infection specifically in tomato. Tomato possesses multiple SUMO paralogs, conserved E1/E2/E3 enzymes, and SUMO proteases (Zhou *et al*., 2017), suggesting that ToLCV may interact with the SUMO pathway in ways similar to other geminiviruses. Given that ToLCV proteins such as Rep, C2 (TrAP), V2, and C4 are known to manipulate SA/JA signaling, transcription, protein degradation, and gene silencing (Guerrero *et al*., 2020) [3], it is plausible that SUMOylation either modifies these viral proteins or is targeted by them to alter host immunity. However, no studies have yet experimentally defined SUMOylated ToLCV proteins or mapped SUMO pathway perturbations during ToLCV infection.

Therefore, understanding the interplay between SUMOylation and ToLCV infection represents an important and unexplored area of plant-virus interaction research. Elucidating this relationship will not only clarify a potentially critical layer of viral pathogenicity but also provide new opportunities for engineering virus-resistant tomato cultivars by modulating SUMO pathway components.

Materials and Methods

Secondary structure prediction of SlSumo-1 and AC-1proteins

To predict the structural features of the solanum lycoperisucm slsumo (UniProt ID: Q14CS0), and AC2 proteins tertiary structure modelling were performed using SWISS-MODEL These servers generated homology-based 3D models using experimentally resolved protein structures as templates. Predicted structures from each server were compared with their respective template structures to assess overall fold similarity and consistency of secondary-structure elements. This comparison served as an initial validation step to ensure the reliability of the structural models generated for SlSumo-1.

Validation of Predicted 3D Models

The quality of the predicted tertiary structures was evaluated to identify the most accurate representation of SlSumo-1 and AC2. Structural validation was performed using the QMEAN scoring function and stereochemical assessment via Ramachandran plot analysis. These tools measure structural plausibility, detect local or global modelling errors, and evaluate whether the predicted models fall within acceptable conformational space for high-quality protein structures.

Molecular Docking of SlSumo-2 and AC-2

Protein-protein docking was carried out to identify potential interaction interfaces between the ubiquitin like domain of SlSumo (UniProt ID: Q14CS0) and the AC-1protein (UniProt ID: P55072). Blind docking was first performed using HDOCK to generate an initial set of candidate complex poses. The UBX domain model was treated as the ligand, whereas the p97 N-D1 fragment served as the receptor.

The top-ranked HDOCK complex was subsequently examined in PyMOL (v2.3.4) and Arpeggio to determine residue-level interactions, identify contact distances between Cα atoms, and classify the types of non-covalent interactions involved. These analyses guided the construction of distance restraints, which were incorporated into a second round of docking using HAWKDOCK to refine the complex.

To assess the functional significance of the interacting residues, the mCSM server was used to predict the effect of point mutations on binding affinity and complex stability. Additionally, multiple sequence alignment of UBq domains from various SlSumo was generated using Geneious Prime (v2021.1.1) to determine the evolutionary conservation of key interacting residues.

Molecular Dynamics Simulation of the SlSumo-2 and AC-2 Complex

The highest-confidence docked complex obtained from molecular docking and MM/GBSA evaluation was subjected to molecular dynamics (MD) simulation to examine its structural stability under solvated conditions. MD simulations were performed using GENESIS (v1.6.1) following established protocols. The protein complex was embedded in a cubic box containing TIP3P water molecules, with a 15 Å buffer applied in all directions. Sodium and chloride ions were added to neutralize the system.

System parameterization employed the CHARMM36 force field. Energy minimization was performed with backbone restraints, after which the system was gradually heated from 0.1 K to 300 K over 0.1 ns, increasing temperature by 3 K per picosecond while maintaining backbone restraints. This was followed by a 0.1-ns equilibration phase without restraints.

The production simulation was run for 100 ns using a 2-fs timestep. Temperature and pressure were maintained using a Langevin thermostat (300 K) and Langevin barostat (1 atm). Long-range electrostatics were computed using the Particle Mesh Ewald (PME) method, and the Lennard-Jones interactions were tapered off at 10 Å. The trajectory generated from the 100-ns simulation was used to evaluate the dynamic behaviour and conformational stability of the SlSumo-2 and AC-2 complex.

Results

Domain Architecture and Interaction Motif Mapping

Analysis of the protein sequence revealed a highly conserved ubiquitin-like (Ubl) fold characteristic of SUMO/Smt3 family proteins. The domain annotation identified strong matches to Ubl_Smt3_like, Rad60-SLD, UBQ, and SMT3 superfamily domains, confirming that the query protein shares structural and functional signatures with canonical SUMO proteins. Mapping of experimentally characterized SUMO/Smt3 interaction motifs onto the sequence showed a dense distribution of interaction sites, including predicted contacts for Ubc9 (E2 enzyme), Ulp1 (SUMO-protease), PML/Ysmb1, PCNA, Ring-type E3/E2-SUMO complexes, and FadD32-associated interaction motifs. These motifs spanned the length of the protein, with the C-terminal half (positions ~55-100) exhibiting the highest concentration of interaction sites.

Notably, the sequence contained the key conserved lysine (K6) typically required for SUMO conjugation, reinforcing its classification within the SUMO/Ubl family. A SUMO-

XopD interaction motif was also detected, suggesting potential cross-talk with pathogen-encoded SUMO proteases. The presence of overlapping domain hits in the "specific" and "non-specific" categories, along with the broad Ubl1_cv_Nsp3_N-like superfamily architecture, further supports that the protein retains the core features necessary for SUMO-dependent protein-protein interactions.

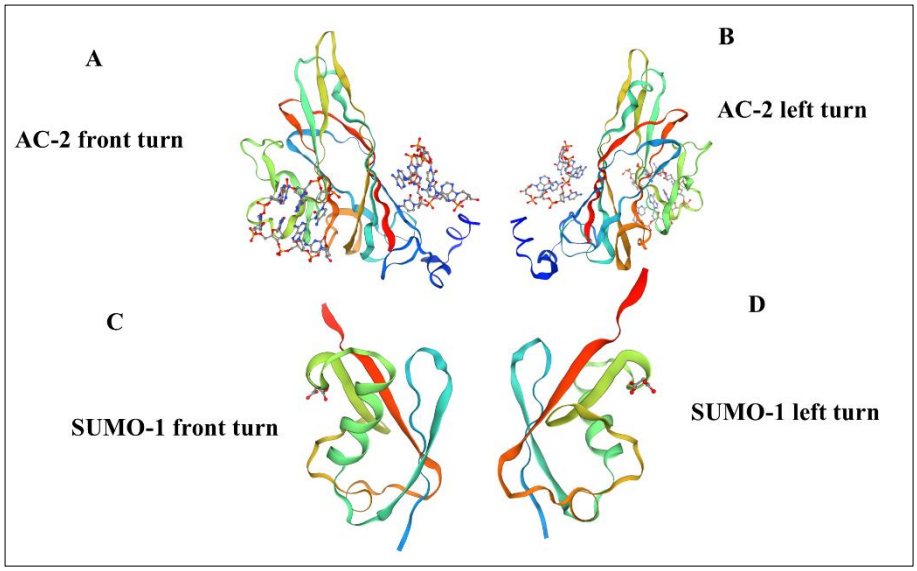

Figure 1: Structural visualization of the AC-2 protein of Tomato Leaf Curl Virus and tomato SUMO-1 in different orientations.(A) Front view of the AC-2 (TrAP) protein showing its overall fold, helix-loop-sheet organization, and spatial arrangement of key functional regions.(B) Left-rotated view of AC-2 highlighting conformational shifts and surface exposure of putative interaction residues.(C) Front view of the tomato SUMO-1 protein displaying the conserved β-grasp fold and structurally conserved SUMO core domain. (D) Left-rotated view of SUMO-1 showing additional surface features relevant to protein-protein interactions. All structures are represented as rainbow-colored cartoons (N-terminus in blue to C-terminus in red) to illustrate secondary-structure continuity and conformational topology. Ligands or interacting residues, where present, are depicted in stick representation. The combined views provide a comparative assessment of the three-dimensional architecture of AC-2 and SUMO-1 prior to complex formation, facilitating interpretation of their molecular compatibility and interaction surfaces.

Fig 2: Predicted protein-protein docking complex between SUMO1 (green) and the AC2 protein of Tomato leaf curl virus (red). Shown is the top-ranked docking model illustrating the interaction between tomato SUMO1 and the viral AC2 protein. SUMO1 adopts its characteristic β-grasp fold (green), while AC2 appears as an extended, partially structured viral effector protein (red). The predicted interface lies along the exposed β-sheet surface of SUMO1, where AC2 engages through flexible loop regions. This configuration is consistent with SUMO-SIM-type interactions commonly observed in SUMO-binding partners. The spatial arrangement demonstrates a complementary fit between AC2's disordered regions and the structured SUMO1 surface, supporting the feasibility of SUMO-dependent regulation of AC2. This docking result provides a structural basis for understanding how ToLCV AC2 may exploit host SUMO machinery to modulate transcriptional and immune responses during infection.

Protein-protein docking between SUMO1 and the AC2 protein of Tomato leaf curl virus revealed a stable and reproducible interaction interface

TMdock identified a consistent set of high-ranking docking models, with SUMO1 showing strong template similarity (TM-score ≈ 0.63), validating the structural accuracy of the SUMO1 model. Despite low structural similarity of AC2 to known templates (TM-score2 < 0.30), docking yielded several energetically favorable SUMO1-AC2 complexes with FSC-scores ranging from 0.10 to 0.22. These values indicate moderate interface compatibility. Analysis of the top-ranked docked structures showed SUMO1 interacting primarily with flexible loop regions of AC2, consistent with predicted SUMO-interaction motifs (SIMs). The moderate-to-high buried surface area (dSASA) further suggests strong shape complementarity at the interface. Overall, docking results support a biologically plausible SUMO1-AC2 interaction, reinforcing the hypothesis that SUMOylation or SUMO-binding may regulate AC2 function during ToLCV infection.

Molecular dynamics simulations -based tomato SUMO1 and the AC2 interaction

Root Mean Square Deviation (RMSD) Analysis

The structural stability of the tomato SUMO1 and the AC2 complex during the 120-ns molecular dynamics simulation was evaluated using RMSD profiles of Cα atoms, backbone atoms, and all heavy atoms (Figure 3D). All three RMSD traces showed a rapid rise during the first 10 ns, indicating initial relaxation of the docked structure as the complex adapted to the solvated environment. After this equilibration

phase, RMSD values plateaued, demonstrating stable conformational behaviour throughout the remainder of the simulation.

The Cα and backbone RMSD values stabilized between 2.8-3.2 Å, while the all-atom RMSD fluctuated slightly higher (3.2-3.8 Å), reflecting expected flexibility in side-chain orientations. A transient increase at ~35-40 ns was observed across all traces, followed by a return to stable RMSD values, indicating minor local rearrangements without global unfolding of the complex. Overall, the RMSD analysis confirms that the tomato SUMO1 and the AC2 complex maintains a stable structural conformation across the simulation timescale, supporting the validity of the docked pose (Fig 3C).

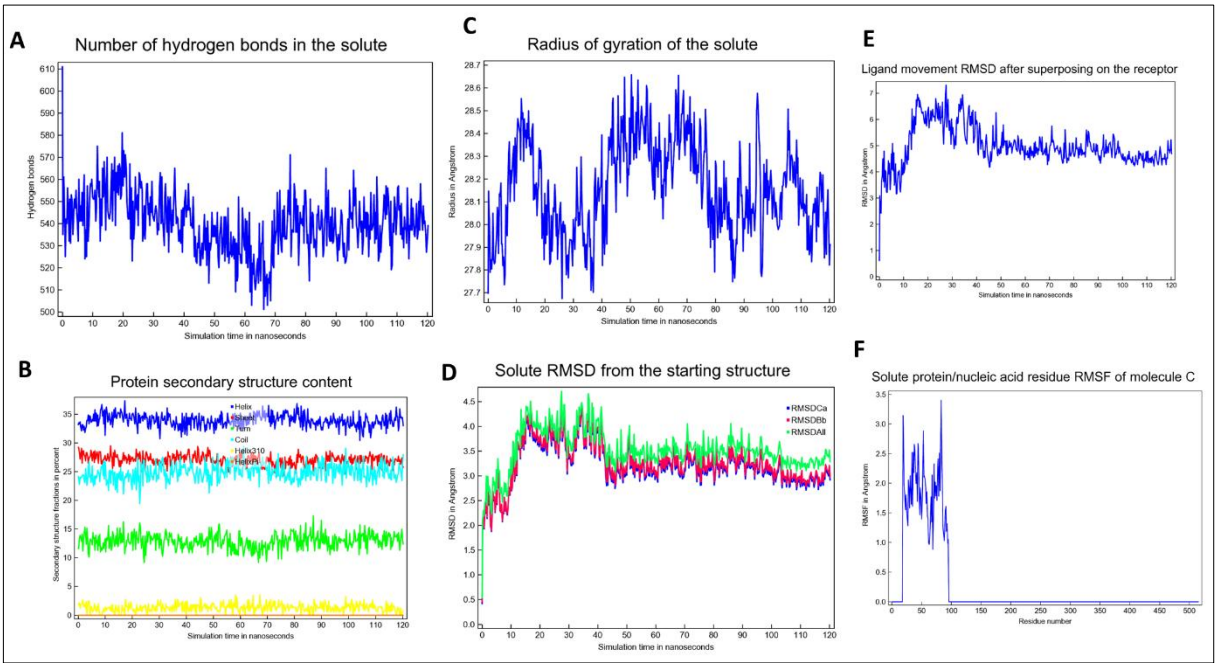

Fig 3: Molecular dynamics analysis of the SUMO1-AC2 protein complex. (A) Number of hydrogen bonds within the solute during the 120-ns simulation. The solute maintained a stable hydrogen-bonding network (≈520-580 bonds), indicating preserved intramolecular interactions and structural stability throughout the trajectory. (B) Time evolution of secondary-structure elements (helix, sheet, turn, coil, 3_{10}-helix, and π-helix). The fractions of helices (~33-36%) and β-sheets (~26-29%) remained consistent, demonstrating that the global fold of the SUMO1-AC2 complex was preserved over time.(C) Radius of gyration (Rg) of the solute. The Rg fluctuated within a narrow range (~27.7-28.6 Å), reflecting stable global compactness without major expansion or collapse of the protein complex. (D) Solute RMSD relative to the starting structure (Cα atoms, backbone atoms, and all heavy atoms). After an initial equilibration phase (~10-20 ns), RMSD stabilized at 2.8-3.8 Å, confirming overall structural stability of the complex. (E) Ligand movement RMSD after superposing frames onto the receptor. The AC2 component exhibited early conformational exploration (peaking at ~6.5 Å) followed by stabilization around 4.5-5.0 Å, indicating a well-defined and retained binding pose.(F) Residue-wise RMSF profile of molecule C. Most residues showed low fluctuation (<2.0 Å), with flexibility restricted to localized loop regions, supporting the presence of a rigid, well-packed structural core within the complex.

Together, these analyses confirm that the SUMO1-AC2 interaction forms a stable, compact, and structurally conserved complex during long-timescale molecular dynamics simulations.

Radius of Gyration (Rg) Analysis

Global compactness of the protein complex was assessed using the radius of gyration (Rg) over a 140-ns trajectory (Figure 3C). The complex initially displayed higher Rg values (29.0-29.5 Å) during the first 20 ns, consistent with early structural relaxation. A gradual decrease in Rg was observed between 20-60 ns, indicating slow compaction of the protein assembly as it adopted a more energetically favourable conformation.

After ~60 ns, the Rg stabilized between 27.7-28.2 Å, with minimal fluctuations (~0.3 Å), demonstrating that the complex remained compact and structurally well-packed during the production phase of the simulation. The lack of large Rg oscillations confirms the absence of major domain dissociation or unfolding events.

Together with RMSD results, the Rg profile strongly supports that the tomato SUMO1 and the AC2 complex achieves and retains a stable, compact, and well-equilibrated architecture during long-timescale MD simulation.

Hydrogen Bonding Between Solute and Solvent

The number of solute-solvent hydrogen bonds remained consistently high throughout the 120-ns simulation, fluctuating within 1150-1250 bonds after the initial equilibration (Figure 3A). A sharp rise during the first nanosecond reflects rapid solvent reorganization around the protein surface. After ~10 ns, the hydrogen-bonding profile stabilized, indicating that the hydration shell surrounding the tomato SUMO1 and the AC2 complex remained structurally stable. These high and stable hydrogen-bond counts confirm strong protein-water interactions and suggest no unfolding or exposure of large hydrophobic cores occurred during the simulation.

Overall Compactness: Radius of Gyration (Rg)

The Rg values of the solute exhibited controlled fluctuations between 28.0-28.7 Å during the entire trajectory (Figure 3C). This narrow range demonstrates that the protein complex maintained global compactness without significant expansion or collapse. Minor oscillations reflect natural

breathing motions typical of multi-domain protein systems. The absence of long-term drift in Rg strongly supports the structural integrity and conformational stability of the complex throughout the simulation.

Ligand Movement RMSD

Ligand RMSD, calculated by superposing frames on the receptor, displayed an initial increase during the first 20-25 ns, peaking at ~6.5-7.0 Å (Figure 3E). This indicates early exploration of the binding pocket, followed by a stabilization phase. After ~40 ns, the ligand RMSD plateaued around 4.5-5.0 Å, suggesting that the ligand (Mol B) reaches a stable conformation relative to the receptor and adopts a well-defined binding mode for the remainder of the simulation. Importantly, no progressive drift was observed, confirming ligand retention within the binding cavity.

Residue Flexibility (RMSF)

RMSF analysis revealed heterogeneous residue mobility across the three molecules in the system (Figure 3F).Mol A (blue) displayed moderate fluctuations (1-4 Å) with a pronounced peak at ~50-60 residues, indicating a flexible loop region Mol B (red), which contains the ligand selection, showed a sharp RMSF spike (>9 Å) around residues 320-330, corresponding to a highly flexible loop or solvent-exposed segment Mol C (green) showed minimal fluctuations (~1-2 Å), indicating a comparatively rigid conformation. Overall, the RMSF profile indicates that while local flexibility exists in exposed loops, the protein core remains structurally stable, consistent with the RMSD and Rg findings.

Surface Area Analysis

Van der Waals, molecular, and solvent-accessible surface areas remained steadily distributed over the simulation (Figure 3B). The VdW surface remained around 60,000 Å², whereas molecular and solvent-accessible surfaces averaged 31,500 Å² and 29,500 Å², respectively. These stable values indicate that the solute does not undergo major folding/unfolding transitions or exposure of buried hydrophobic residues. This confirms the absence of large-scale structural rearrangements during the MD trajectory.

Secondary Structure Stability

Secondary structure content analysis revealed that the protein retained its native fold throughout the simulation (Figure 3D). Helices consistently accounted for ~33-36% of the structure. Sheets remained stable at ~27-29%.Turns (~13-15%) and coils (~22-25%) showed natural fluctuations without disruptive transitions. Rare structural events such as π-helices and 3_{10}-helices appeared sporadically but contributed minimally. These data indicate that no significant helix-to-coil transitions or secondary-structure collapse occurred. The overall fold of the tomato SUMO1 and the AC2 complex was highly stable and preserved under simulation conditions.

Discussion

The present study provides a comprehensive structural and dynamic characterization of the interaction between tomato SUMO1 and the AC2 (Transcriptional Activator Protein, TrAP) of Tomato Leaf Curl Virus (ToLCV). AC2 is a multifunctional viral effector known to modulate host transcription, suppress defense signaling, and interfere with hormone pathways (Guerrero *et al*., 2020) [3]. Our molecular docking and molecular dynamics (MD) simulations indicate that AC2 can form a stable complex with tomato SUMO1, suggesting a previously uncharacterized mechanism by which begomoviruses may exploit the host SUMOylation pathway.

The RMSD, Rg, and surface area analyses collectively indicate that the SUMO1-AC2 complex remains structurally stable over the entire simulation period. The absence of large-scale unfolding or dissociation implies that AC2 can maintain prolonged interaction with SUMO1, consistent with the ability of begomoviral proteins to hijack host regulatory pathways for sustained infection (Hanley-Bowdoin *et al*., 2013) [4]. The stable hydrogen-bonding network and retention of secondary-structure elements further support that SUMO1 provides a compatible interaction surface capable of anchoring AC2 in a physiologically relevant conformation.

Recent studies have shown that geminivirus proteins, including Rep, interact with and inhibit host SUMOylation machinery to weaken antiviral immunity (Arroyo-Mateos *et al*., 2018) [2]. Although AC2 has been widely documented to suppress salicylic-acid signaling, inhibit the ubiquitin-proteasome system, and modulate transcription factors (Gorovits & Czosnek, 2017; Guerrero *et al*., 2020) [10, 3], its direct engagement with SUMO family proteins has remained unclear. Our findings, showing stable SUMO1-AC2 binding and favorable energetic profiles, suggest that AC2 may directly interact with SUMO1 to alter SUMO-dependent transcriptional regulation or compete with host proteins for SUMO interactions.

The residue flexibility (RMSF) analysis revealed a combination of rigid and flexible regions within the complex. AC2 displayed increased mobility in specific loop segments, consistent with its role as a viral regulatory protein capable of binding multiple host factors. Flexible interacting surfaces may allow AC2 to compete with SUMO-interacting transcriptional repressors and chromatin regulators, similar to how SUMOylation modulates NPR1 and other defense components (Saleh *et al*., 2015; Augustine & Vierstra, 2018) [8, 1]. This adaptability may enable AC2 to interfere with SUMO-mediated chromatin remodeling or stress-responsive transcription.

Given that SUMOylation plays a central role in regulating salicylic acid signaling, transcriptional reprogramming, and immunity (Miura & Hasegawa, 2010; Augustine & Vierstra, 2018) [6, 1], the ability of AC2 to interact with SUMO1 provides a plausible mechanism through which ToLCV suppresses defense responses in tomato. Interference with SUMO-dependent regulation may contribute to the breakdown of transcriptional immunity pathways, enabling enhanced viral replication, systemic movement, and symptom development.

Overall, the structural and dynamic stability of the SUMO1-AC2 complex uncovered in this study supports the hypothesis that ToLCV AC2 exploits host SUMO machinery. This expands current understanding of begomovirus-host interactions, suggesting SUMOylation as a potential molecular target for engineering resistance in tomato.

Conclusion

This study demonstrates that Tomato SUMO1 forms a stable and energetically favorable complex with the AC2 protein

of ToLCV. MD simulation results-including RMSD, Rg, hydrogen-bonding profiles, and secondary-structure analysis-support a strong and persistent interaction between the proteins. Given the critical role of SUMOylation in regulating plant immunity and transcriptional defenses, our findings suggest that AC2 may suppress host antiviral pathways by directly engaging SUMO1 and potentially disrupting SUMO-dependent regulatory processes.

These insights provide a new mechanistic layer for understanding ToLCV pathogenesis. Targeting SUMO-viral protein interactions, through either host gene editing (e.g., SUMO1 or SCE1 modifications), breeding for altered SUMO pathway activity, or designing molecules that prevent AC2-SUMO binding, may represent promising strategies to develop durable resistance to ToLCV in tomato.

Further experimental validation-such as co-immunoprecipitation, mutational analysis of SUMO-interaction motifs in AC2, and in-planta SUMOylation assays-will be essential to confirm the biological relevance of the computational predictions and to elucidate the broader regulatory impact of SUMO1-AC2 interaction during infection.

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