SlmA Protein: Master Coordinator of Bacterial Chromosome Partitioning and Cell Division – An Extensive Review

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Abstract
The bacterial life cycle dependently requires precise coordination between chromosomal duplication and binary fission because any disruption will create cells that either lack DNA or possess genetic defects. SlmA protein which E. coli and Gram-negative bacteria use as their main controller of nucleoid occlusion system operates as a spatial control system that prevents Z-ring development on bacterial chromosomes. The complete review presents research findings about SlmA which researchers collected during two decades starting from its original discovery until they discovered its molecular functions. We explore the structural basis of SlmA-DNA binding, its unique distribution across chromosomal macrodomains, and its direct interaction with the key division protein FtsZ. Particular emphasis is placed on recent paradigm-shifting discoveries, including SlmA's role in forming dynamic biomolecular condensates via phase separation, its newly discovered membrane-binding capabilities, and its functional balance with positive regulators like ZapA. We also examine SlmA's important function in bacterial stress recovery, particularly during filamentation reversal. Finally, we discuss the therapeutic potential of targeting the SlmA-FtsZ-SBS pathway, with special attention to recent advances in Benzodioxane-benzamide compounds and their novel mechanism of action. This review integrates classical findings with the most recent literature (2022-2025) to provide a total picture of how this remarkable protein ensures faithful chromosome inheritance across bacterial generations.

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1. Introduction

The bacterial cell division is an intricately regulated process that requires precise execution, both spatially and temporally. However, at its core, this bacterial cell division process is mediated by a set of conserved proteins, such as FtsZ, which is homologous to tubulin and forms a dynamic structure beneath the bacterial cell membrane (Bi & Lutkenhaus, 1991). The Z-ring structure attracts more than 30 different proteins which create the divisome complex that enables cell division into two daughter cells (den Blaauwen *et al*.,2017) [6].

Bacteria that divide face their main obstacle because they must establish their Z-ring at midcell positions after their chromosomes complete both replication and segregation processes. Premature division or division over incompletely segregated chromosomes can lead to guillotining of the nucleoid which produces anucleate cells and cells with damaged DNA that are. The challenge is focused towards the high documentation of the laws of division inviable (Bernhardt 2012) [2]..

In E. coli, three major systems cooperate to ensure accurate division site placement: 1- The Min System: This mechanism prevents Z-ring formation at the cell poles due to the oscillatory behavior of MinC, MinD, and MinE proteins (Raskin & de Boer, 1999) [15].

2- Nucleoid Occlusion (NO): This mechanism prevents Z-ring formation at the nucleoid, and SlmA is known to be the major

player (Bernhardt & de Boer, 2005) [1].
3-Ter Linkage: This mechanism is known to positively regulate cell division at midcell by MatP-mediated Ter macrodomain anchoring (Mercier *et al*., 2008) [12].
This review focuses on the nucleoid occlusion mediator SlmA, tracing its journey from a genetic curiosity to one of the best-understood bacterial division regulators, and underscoring the latest insights that further to redefine our knowledge of its underlying mechanism.

2. Discovery and Initial Characterization of SlmA

2.1. Genetic Identification of a Novel Division Regulator

The study of SlmA began with a traditional genetic screen for synthetic lethal interactions. (Bernhardt & de Boer 2005) [1] hypothesized that if the Min system is responsible for preventing polar division, there must be another system that prevents division across the nucleoid, and that a cell with both systems inactivated would be lethal. They searched for mutations that would be lethal in a Δmin strain and isolated a new gene, which they called slmA (synthetic lethal with a defective min system A). The analysis showed that slmA min double mutants exhibited a distinct phenotypic pattern. The cells produced extended filaments which contained multiple Z-rings that assembled at incorrect sites, including their direct overlap with nucleoids. Time-lapse microscopy showed that the misplaced division events produced DNA guillotining which resulted in anucleate cell production, which explained the synthetic lethality. The slmA single mutants showed only minor division problems under normal laboratory conditions, which demonstrated that they functionally duplicated the Min system operational abilities.

2.2. Early Biochemical Characterization

Scientists discovered through their first biochemical tests that SlmA operates as a DNA-binding protein which belongs to the TetR transcriptional regulation protein family. The research showed that SlmA exists as stable dimers in solution while its structure includes an N-terminal helix-turn-helix DNA-binding domain together with a C-terminal dimerization domain. The research demonstrated that SlmA binds directly to FtsZ which leads to the hypothesis that SlmA controls FtsZ polymerization onto the nucleoid through its inhibitory action. The overproduction of slmA results in complete division blockage which causes wild-type cells to develop into filamentous structures. The SlmA DNA-binding function proved essential since helix-turn-helix domain mutations blocked both DNA binding and division inhibition (Bernhardt & de Boer, 2005) [1]. The early research findings established the primary model which shows that SlmA functions as a DNA-binding protein that prevents FtsZ from forming assembly through its binding to chromosomes.

3. Molecular and Structural Basis of SlmA Function

3.1. Genome-wide Identification of SlmA Binding Sites

In 2011 two research teams effectively used the ChIP-Seq method which combines chromatin immunoprecipitation with high-throughput sequencing to determine SlmA binding sites throughout the entire E. coli chromosome. The results showed that SlmA interacts with a distinct 12-base pair palindromic sequence: 5'GTGAGTACTCAC3'. The chromosome contains approximately 50 sites which scientists designate as SlmA Binding Sites. The main finding showed that the sites occurred in two different patterns which resulted in high site concentration near the Ori macrodomain and complete absence of sites near the Ter macrodomain. The functional implications of this uneven distribution are significant, since as chromosomes replicate and separate, the Ter area, which does not contain SBS sites, is the last to depart from the midcell area, thereby establishing an SBS-free area where it should be—during cell division itself (Cho *et al*., 2011) [5].

3.2. Structural Insights into SlmA-DNA-FtsZ Interactions

The research team conducted a structural analysis of SlmA crystal to uncover its distinctive properties. (Tonthat *et al*., 2011) [18] determined the crystal structure of SlmA dimer at a resolution of 2.5 Å, and it was found that SlmA contains a typical TetR fold with some unique features. The DNA-binding helices work together to recognize the palindromic SBS sequence while the C-terminal domain functions as a dimerization and FtsZ interaction partner. The research team conducted additional small-angle X-ray scattering experiments to study SlmA-DNA complexes, which resulted in new structural findings about these complexes. Tonthat *et al*. revealed that two SlmA protein dimers cooperate to bind to one SBS site, forming a tetrameric structure, or "dimer of dimers." The tetrameric structure can bind to DNA through four dimers that attach to regions beyond the central SBS site. The tetrameric SlmA-DNA structure has four surfaces that can interact with FtsZ, which could hold FtsZ filaments in a non-productive structure.

3.3. Mechanism of FtsZ Inhibition

The present model for DNA-based SlmA inhibition shows that FtsZ polymerization process is disrupted by the existing mechanism. The binding of SlmA to DNA fails to impact FtsZ assembly process and GTPase activity at all times. When SlmA attaches to SBS DNA, it acquires the power to obstruct FtsZ polymerization according to (Cho *et al*. 2011) [5] research.
The process of inhibition requires both physical sequestration and architectural disruption to function. The SlmA-DNA tetramer interacts with the conserved C-terminal tail of FtsZ, which is important for the interaction of FtsZ with many other regulatory proteins. The interaction between the two parties allows FtsZ to create short protofilaments but stops the protofilaments from developing into the Z-ring necessary higher-order structure. Electron microscopy has been used to show that, when SlmA-SBS complexes are present, FtsZ creates spiral structures instead of the long filaments that form under permissive conditions (Tonthat *et al*., 2013) [19].

4. New Horizons: Biomolecular Condensates and SlmA Dynamics

4.1. Phase Separation and Reversible Condensate Formation

The latest promising development in SlmA research shows that SlmA, FtsZ, and SBS DNA can interact to create dynamic biomolecular condensates through liquid-liquid phase separation. The study by Monterroso and his colleagues showed that FtsZ and SlmA and SBS DNA form reversible phase separation under cytomimetic conditions which simulate bacterial cytoplasm macromolecular crowding. The system produces spherical droplets which can merge together and display liquid-like properties.
These condensates are highly dynamic and responsive to nucleotide state. The addition of GTP triggers rapid

reorganization of condensates into organized FtsZ polymers, while subsequent GTP hydrolysis leads to polymer disassembly and re-formation of condensates. This sets up a reversible cycle between a compacted and assembled state where SlmA-SBS complexes act as the modulators of this switching operation The functional significance of this phase behavior exhibits multiple dimensions. Condensates function as temporary storage spaces which hold inactive GDP-bound FtsZ until the cells require division signals. The structures operate as stress-response systems which shield FtsZ from destruction and improper contact during hostile environments. The condensates establish under conditions which exist in the body which demonstrates their status as actual components of cell organization rather than laboratory-based artifacts (Monterroso *et al*., 2023) [13].

4.2. The ZapA-SlmA Balance: Tuning FtsZ Fate

SlmA promotes the creation of condensates while blocking the process of polymerization and this effect works against the function of ZapA which serves as a positive regulatory element. The FtsZ-binding protein ZapA functions as a well-researched component because it maintains FtsZ polymers and enables their lateral aggregation into bundles (Gueiros-Filho & Losick, 2002) [7].

(Monterroso *et al*., 2023) [13]. conducted a dedicated examination to study how SlmA and ZapA interact in competitive situations. The two regulators establish non-competitive binding to FtsZ because they can bind to the same FtsZ molecules at the same time. The outcome—whether FtsZ forms stable polymers or partitions into condensates—depends on the ratio of ZapA to SlmA. The crowded cytoplasm conditions establish an advantage for ZapA which leads to increased polymer stability through the existence of SlmA-SBS complexes.. This finding demondtrate that division site selection is not simply a matter of SlmA-mediated inhibition but involves a dynamic equilibrium between opposing regulatory forces. The local concentration of positive and negative regulators, which may vary across different cellular compartments, determines the propensity for Z-ring assembly.

4.3. SlmA-Membrane Interactions: A New Dimension

For many years, the SlmA field operated under the assumption that SlmA's actions were confined to the nucleoid surface. A groundbreaking study by Paccione *et al*. 2022 showed that SlmA directly binds to membrane lipids which proved the existing scientific view wrong.

The researchers demonstrated that fluorescently labeled SlmA directly binds to lipid surfaces when using supported lipid bilayers and giant unilamellar vesicles as their model membrane systems. The interaction demonstrates two distinct elements because both electrostatic and hydrophobic forces affect its strength. The interaction becomes stronger because of electrostatic effects at reduced ionic strength but it maintains binding to neutral lipids and shows partial response to high salt because of hydrophobic forces. The membrane interaction has deep effects on SlmA function. The presence of lipid surfaces with glutamate anions, which serve as major cytoplasmic ions, leads to increased formation of SlmA-SBS-FtsZ condensates that move to the membrane. The condensates exhibit dynamic behavior, as they merge together to increase their size, while specifically drawing in GDP-bound FtsZ from their external surroundings. The membrane-binding ability of SlmA indicates that it not only blocks FtsZ assembly at the nucleoid but also directs FtsZ to store in membrane-bound areas during times when division should not occur. The research introduces a completely new element to our comprehension of bacterial cell spatial control mechanisms (Paccione *et al*. 2022) [14].

5. SlmA in Stress Response and Filament Recovery 5.1. Filamentation as a Stress Response

Bacteria show a filamentous growth phenotype when they face three types of stressful situations which include DNA damage and replication problems and exposure to antibiotics. The process of filamentation causes cells to grow continuously because they do not undergo division, which results in the creation of multinucleate filaments that can extend up to hundreds of microns. The response allows DNA repair and stress resolution activities to take place before the organism decides to start dividing. The filaments need to undergo fast and accurate division process after stress condition has ended because this process helps return the cells to their normal shape. The recovery process presents two main difficulties, which involve understanding how cells manage several division processes that happen on one filament while making sure that each division septum develops between separated chromosomes (Justice *et al*., 2008) [11]..

5.2. SlmA's Role in Filament Recovery

A detailed study conducted by (Cayron *et al*., 2023) [4]. used microfluidics and time-lapse microscopy to observe the repair of E. coli filaments following stress relief. The results obtained from the study showed the different roles played by the Min system and SlmA. The Min system is primarily involved in positioning division sites at the poles of filaments and between nucleoids. In Min cells, filaments frequently begin divisions from improper sites, such as over nucleoids. However, SlmA plays a distinct and critical role in controlling the timing of division. In slmA filaments, divisions occur prematurely, before chromosome segregation is complete, leading to DNA guillotining and production of inviable cells). This timing function appears to involve SlmA-mediated monitoring of chromosome segregation status. SlmA-SBS complexes on segregating chromosomes create a dynamic inhibitory landscape that only clears completely when chromosomes have fully separated. This ensures that septation does not commence until the last link between segregating chromosomes is broken. (Cayron *et al*., 2023) [4].

5.3. Synchronization with Other Systems

The process of filament recovery requires coordination with both Ter macrodomain-binding protein MatP and DNA translocase FtsK. The midcell attachment of Ter region occurs through MatP which functions as the tethering mechanism and FtsK operates as the DNA pumping system that moves DNA through the septum for complete segregation (Stouf *et al*., 2013) [17]. SlmA works together with these systems to create another protective system which stops cells from entering the process of division. Filament recovery happens through a process of rapid asymmetric divisions which differ from multiple simultaneous divisions

at different locations. The pattern enables cells to reach their normal size more quickly while their survival remains intact. The SlmA protein controls division timing which serves as a critical component for executing the recovery operation (Cayron *et al*., 2023) [4].

6. Therapeutic Targeting of the SlmA-FtsZ-SBS Pathway

6.1. FtsZ as an Antibiotic Target

The problem of antibiotic resistance has created an imperative need to develop new antimicrobial agents with novel modes of action. FtsZ has proven to be an attractive target because it is essential, highly conserved among bacteria, and sufficiently different from eukaryotic tubulin to be selectively inhibited (Haranahalli *et al*., 2020) [8].

Scientists have created multiple types of FtsZ inhibitors which include benzamides and taxanes and berberines to specifically block FtsZ proteins. The benzodioxane-benzamide (BDOB) derivatives from this FtsZ inhibitor classification demonstrate exceptional abilities to fight against various Gram-positive and Gram-negative bacterial strains including methicillin-resistant Staphylococcus aureus (MRSA) and vancomycin-resistant bacteria.

6.2. BDOBs and the SlmA Connection

The researchers established an unexpected relationship between BDOB compounds and SlmA regulatory systems through their October 2025 research study which took place at the International Journal of Biological Macromolecules. In their study, the researchers sought to understand the effects of three potent BDOB derivatives, FZ95, FZ100, and FZ116, on FtsZ dynamics in the presence of SlmA-SBS complexes. The results showed that these compounds caused FtsZ polymers to stay intact against SlmA-SBS disassembly. The compounds created FtsZ protection against SlA. The BDOB compounds create FtsZ condensates and polymer states because they block FtsZ from switching between these two states. The antibacterial mechanism depends on this disruption as an essential element. The compounds disrupt the FtsZ state equilibrium which leads to a SlmA (Sobrinos-Sanguino *et al*., 2025) [16] pattern of division time and position that cells cannot control.The study demonstrated that BDOB treatment causes cells to misplace their division processes and their ability to withstand stress double their normal level of stress resistance. The research indicates that using BDOBs together with DNA-damaging agents will create stronger antibacterial effects than either treatment used alone.

6.3. Implications for Drug Development

The results of this research study create possibilities for developing new antibiotics. The next-generation compounds should be developed to target FtsZ regulatory pathways instead of their current function which only stops FtsZ polymerization. The approach provides better selectivity which results in decreased harmful effects. The SlmA-FtsZ-SBS interaction surface functions as a potential drug target according to the discovery. The small molecules which prevent SlmA from binding to FtsZ and disrupt SlmA-DNA binding can shut down the nucleoid occlusion system which makes bacteria more vulnerable to division pressure.

7. Evolutionary Perspectives: Convergent Solutions to a Common Problem

7.1. SlmA in Gram-Negative Bacteria

SlmA exists as a conserved protein throughout various Gram-negative bacterial species which include Escherichia, Salmonella, and Klebsiella together with their related genera. The study of sequence data reveals that essential DNA-binding domain and FtsZ interaction surface residues remain unchanged throughout most species according to research which demonstrates basic mechanism preservation (Bernhardt, 2012) [2]. SBS-like sequences show a conservation pattern since they show greater presence near origins while showing lower presence near terminus regions across different species. The "SBS-free zone" mechanism establishes division site specifications as a common characteristic which all SlmA-containing bacteria exhibit.

7.2. Noc: A Convergent Solution in Gram-Positive Bacteria

Gram-positive bacteria, which include Bacillus subtilis as their main research model, must control their division process to protect their nucleoid structure. The bacteria developed a unique protein which they use to solve this problem. (Noc nucleoid occlusion protein)(Wu & Errington, 2004) [20]. Noc and SlmA operate the same function despite absence of both sequence and structural links between them. They function as DNA-binding proteins which control FtsZ assembly through their specific binding to the chromosome. The two proteins from different evolutionary backgrounds developed identical solutions to one biological challenge. Noc mechanism of action has been revealed through recent structural investigations. (Jalal *et al*., 2023) [10] determined the structure of the Noc-DNA complex. The Noc-DNA complex structure shows that Noc exists as a dimer which employs a different binding method than SlmA. Noc displays the ability to bind CTP and hydrolyze it, which serves as a critical functional requirement for the protein. Noc uses CTPase activity as a timing mechanism which controls nucleoid occlusion throughout the cell cycle, which differs from the control methods that SlmA employs.

7.3. Why Two Different Solutions?

The existence of two different nucleoid occlusion mechanisms introduces intriguing possibilities for evolutionary research. The common ancestor of bacteria must have possessed an alternative system because SlmA and Noc developed through separate processes which occurred after the evolutionary split between Gram-positive and Gram-negative bacteria. The evolution of two separate nucleoid occlusion mechanisms from a single ancestral system indicates that the selective pressure for these mechanisms functioned as an independent evolutionary force.. The different cell biological capabilities of Gram-positive bacteria and Gram-negative bacteria lead to different biological processes which result in these differences. Gram-positive bacteria possess a cell wall structure which lacks an outer membrane and which creates unique limitations on their ability to control cell division. The organisms have different chromosomal replication and segregation systems which depend on particular regulatory mechanisms for their control.

8. Conclusions and Future Perspectives

8.1. Current Understanding

The research conducted over twenty years about SlmA has discovered an intricate regulatory network which governs its functions. The present research shows that SlmA exhibits the following abilities. SlmA: - Binds specific DNA sequences (SBS) which occur at different rates throughout the

chromosome.

• Forms tetrameric complexes on DNA which create different areas for FtsZ to bind.
• The protein inhibits FtsZ polymerization through a DNA-dependent mechanism.
• The protein forms liquid biomolecular condensates through phase separation.
• The protein interacts with lipid membranes which adds extra spatial control to its regulatory mechanisms.
• The protein uses ZapA as a positive regulator to control the assembly process of FtsZ.
• The protein regulates the timing of cell division by monitoring stress recovery and filamentation.
• Certain antibiotics target this protein because they stop its regulatory functions.

8.2. Open Questions

The researchers have made progress but still face several remaining questions. The research team studied how SlmA protein activity changes during different stages of cell development. Scientists discovered that SlmA-SBS complexes disappear from midcell locations when chromosomes begin their segregation process, but researchers have not yet identified the molecular signals which cause this process to occur. The study aims to determine whether SlmA protein actively detaches from DNA or whether it gets moved away through the actions of replication and segregation equipment. Scientists seek to identify all proteins that interact with SlmA. The research study investigates whether SlmA has additional connections to division proteins and replication machinery components beyond its relationship with FtsZ.

The process involves multiple interactions which create additional methods for maintaining operational control. The research team investigates how phase separation mechanisms apply across different situations. The study investigates whether condensate formation serves as a regulatory mechanism for bacterial division through its impact on other bacterial division regulators. The research investigates whether this pattern functions as a fundamental organizational system that exists throughout prokaryotic organisms. The researchers investigate whether doctors can use SlmA pathways as a drug target for therapeutic purposes. The BDOB research results show potential, but scientists need to conduct further medicinal chemistry work and preclinical trials before they can turn these findings into usable clinical antibiotics. The study compares SlmA and Noc systems to determine their distinct operational methods. The research reveals fundamental division control principles, while also showing how this information can create antibiotics that treat both Gram-positive and Gram-negative bacterial infections.

8.3. Future Directions

The new technologies which are currently being developed will provide solutions to the existing problems. Cryo-electron tomography of dividing cells will show researchers the natural structural design of SlmA-SBS-FtsZ complexes when studied through this method. Single-molecule tracking in live cells will show scientists how SlmA creates and breaks its connections with chromosomal sites. The researchers found that *in vitro* reconstitution of minimal division systems allows them to study all parts of the regulatory networks. Researchers will study bacterial cell division patterns through microfluidic devices which enable them to observe cells over extended periods in controlled laboratory settings. The combination of these methods with genetic modification and environmental control methods will help scientists study how SlmA processes different signals to control division accuracy. From a therapeutic perspective, structure-based drug design targeting the SlmA-FtsZ interaction surface could yield novel compounds. The researchers used high-throughput screening to find molecules which break SlmA-DNA binding and discovered new chemical scaffolds. The researchers discovered that combination therapies which attack multiple systems that control division will create stronger antibacterial outcomes while decreasing the chance of resistance development.

References

1. Bernhardt TG, de Boer PAJ. SlmA, a nucleoid-associated, FtsZ binding protein required for blocking septal ring assembly over chromosomes in *Escherichia coli*. Mol Cell. 2005;18(5):555–564.
2. Bernhardt TG. The nucleoid occlusion model of cell division control. Nat Rev Microbiol. 2012;10(3):175–183.
3. Bi E, Lutkenhaus J. FtsZ ring structure associated with division in *Escherichia coli*. Nature. 1991;354(6349):161–164.
4. Cayron J, Dedieu-Berne A, Lesterlin C. Bacterial filaments recover by successive and accelerated asymmetric divisions that allow rapid post-stress cell proliferation. Mol Microbiol. 2023;119(2):237–251.
5. Cho H, McManus HR, Dove SL, Bernhardt TG. Nucleoid occlusion factor SlmA is a DNA-activated FtsZ polymerization antagonist. Proc Natl Acad Sci U S A. 2011;108(9):3773–3778.
6. den Blaauwen T, Hamoen LW, Levin PA. The divisome at 25: the road ahead. Curr Opin Microbiol. 2017;36:85–94.
7. Gueiros-Filho FJ, Losick R. A widely conserved bacterial cell division protein that promotes assembly of the tubulin-like protein FtsZ. Genes Dev. 2002;16(19):2544–2556.
8. Haranahalli K, Tong S, Ojima I. Recent advances in the discovery and development of antibacterial agents targeting the cell-division protein FtsZ. Bioorg Med Chem. 2020;28(1):115238.
9. Haydon DJ, Stokes NR, Ure R, *et al*. An inhibitor of FtsZ with potent and selective anti-staphylococcal activity. Science. 2008;321(5896):1673–1675.
10. Jalal AS, Tran NT, Le TBK. Structural basis of nucleoid occlusion mediated by the *Bacillus subtilis* protein Noc. J Biol Chem. 2023;299(2):102834.
11. Justice SS, Hunstad DA, Cegelski L, Hultgren SJ. Morphological plasticity as a bacterial survival strategy. Nat Rev Microbiol. 2008;6(2):162–168.
12. Mercier R, Petit MA, Schbath S, *et al*. The MatP/matS site-specific system organizes the terminus region of the *E. coli* chromosome into a macrodomain. Cell. 2008;135(3):475–485.
13. Monterroso B, Zorrilla S, Sobrinos-Sanguino M, *et al*. Bacterial division ring stabilizing ZapA versus destabilizing SlmA modulate FtsZ switching between biomolecular condensates and polymers. Open Biol. 2023;13(3):220324.
14. Paccione G, Sobrinos-Sanguino M, Borbolla M, *et al*.

Lipid surfaces and glutamate anions enhance formation of dynamic biomolecular condensates containing bacterial cell division protein FtsZ and its DNA-bound regulator SlmA. Biochemistry. 2022;61(22):2482–2493.
15. Raskin DM, de Boer PA. MinDE-dependent pole-to-pole oscillation of division inhibitor MinC in *Escherichia coli*. J Bacteriol. 1999;181(20):6419–6424.
16. Sobrinos-Sanguino M, Zorrilla S, Monterroso B, *et al*. Benzodioxane-benzamides targeting bacterial cell division protein FtsZ potentially disrupt SlmA-mediated nucleoid occlusion and reversible biomolecular condensation. Int J Biol Macromol. 2025;148516.
17. Stouf M, Meile JC, Cornet F. FtsK actively segregates sister chromosomes in *Escherichia coli*. Proc Natl Acad Sci U S A. 2013;110(27):11157–11162.
18. Tonthat NK, Arold ST, Pickering BF, *et al*. Molecular mechanism by which the nucleoid occlusion factor SlmA keeps cytokinesis in check. EMBO J. 2011;30(1):154–164.
19. Tonthat NK, Milam SL, Chinnam N, *et al*. SlmA forms a higher-order structure on DNA that inhibits cytokinetic Z-ring formation over the nucleoid. Proc Natl Acad Sci U S A. 2013;110(26):10586–10591.
20. Wu LJ, Errington J. Coordination of cell division and chromosome segregation by a nucleoid occlusion protein in *Bacillus subtilis*. Cell. 2004;117(7):915–925

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