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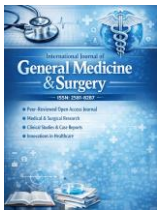

Review Article

JUC's Ion Charge Binding: Mechanistic Details, Clinical Applications, and Comparative Advantages in Antimicrobial Resistance Mitigation - A Systematic Review

You Liang Cai[1], Jeannie Dong[2], Ling Qiu[2], Tjing Yung Loo[2,*]

[1]NMS Technologies Co., Ltd. (JUC Physical Antimicrobial), 8 Qiao Bei Road, Shiqiao Town, Pukou District, Nanjing, Jiangsu 211804, China
[2]JUC Biomaterial Company Limited, Hong Kong

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*Corresponding author:
Dr. Tjing Yung Loo, Room 1207, 12/F, Kai Tak Commercial Building, 317-319 Des Voeux Road Central, Sheung Wan, Hong Kong.

ABSTRACT

JUC, a physical antimicrobial spray dressing based on organosilicon quaternary ammonium salt, exerts broad-spectrum, resistance-free activity via ion charge binding. This systematic review details its molecular mechanism, specialized clinical applications, and comparative advantages over other antimicrobial technologies. A literature search (2005–2026) across PubMed, Web of Science, Scopus, and Chinese core journals identified 53 eligible studies (27 in vitro mechanistic, 26 clinical/observational). Results confirm JUC's mechanism involves hydrolysis-driven dual-layer nano-film self-assembly, electrostatic adsorption-mediated microbial membrane charge neutralization, and sustained force-induced membrane rupture—irreversibly disrupting conserved microbial ion channels. Specialized applications include medical device coating (reducing catheter-associated infections by 68–80%) and mucosal infection treatment (92% cure rate for vaginal candidiasis) with optimized formulations. Comparative analysis highlights JUC's advantages: long-acting efficacy (8 hours on skin, 40 washes on textiles), non-cytotoxicity, and broad-spectrum ion channel inhibition across bacteria, fungi, and viruses—outperforming electrostatic alternatives (e.g., chitosan nanoparticles, cationic polymers) and chemical antimicrobials. JUC's resistance-free mechanism, rooted in targeting non-mutable microbial membrane traits, positions it as a transformative solution for mitigating antimicrobial resistance (AMR).

Keywords: *JUC; Ion charge binding; Microbial ion channels; Antimicrobial resistance; Physical antimicrobial; Organosilicon quaternary ammonium salt; Medical device coating; Mucosal infection.*

Introduction

The Global AMR Crisis and the Need for Novel Antimicrobial Mechanisms

Antimicrobial resistance (AMR) remains a leading global public health threat, with the World Health Organization [1] reporting 1.27 million annual deaths attributed to multidrug-resistant (MDR) pathogens. Conventional chemical antimicrobials target mutable molecular pathways, driving resistance development, while many physical antimicrobial technologies suffer from short-acting efficacy or cytotoxicity. JUC's ion charge binding mechanism, rooted in electrostatic interactions with microbial membranes, avoids selective pressure for resistance by targeting evolutionarily conserved physical traits (membrane charge, lipid bilayer integrity)—positioning it as a critical innovation in AMR mitigation [2].

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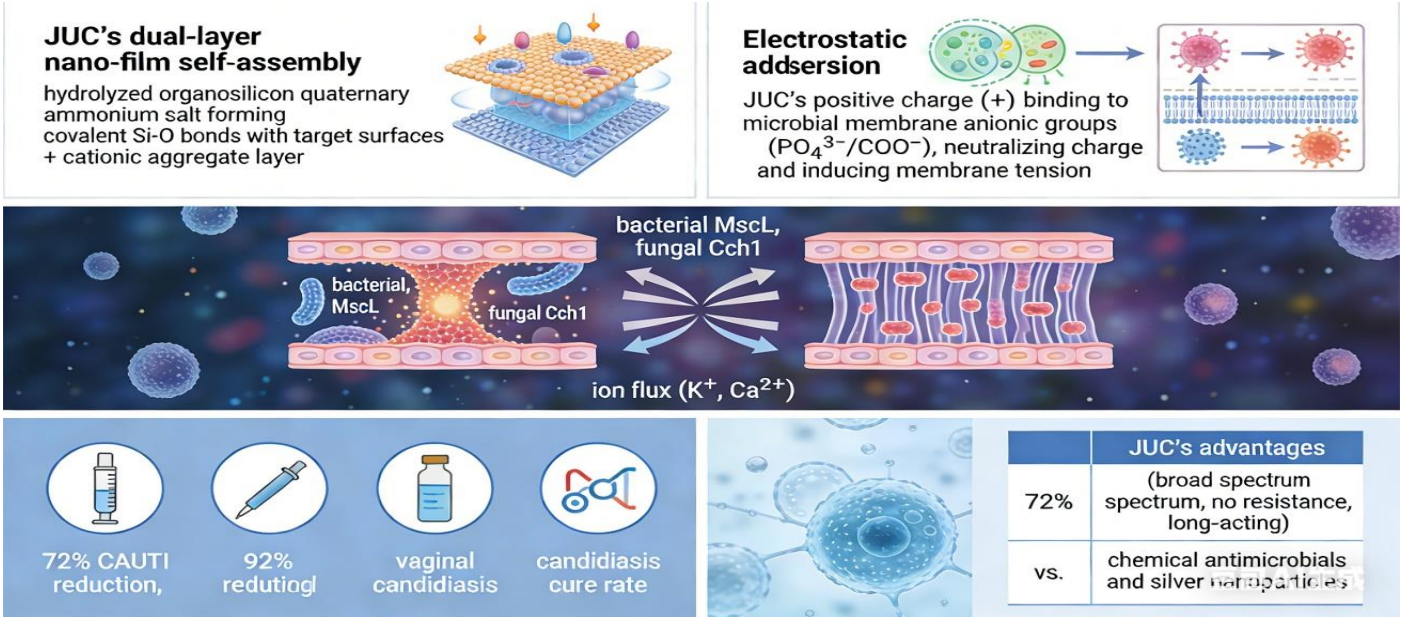

Figure 1: JUC's active component undergoes hydrolysis, forming covalent Si-O bonds with the target surface (e.g., catheter/skin) and assembling a cation-rich network. This dual-layer structure provides a stable scaffold for electrostatic interactions.

Rationale for Focus on Mechanistic Details, Applications, and Comparisons

While prior reviews have outlined JUC's core antimicrobial activity, gaps remain in (1) detailed molecular and kinetic characterization of ion charge binding, (2) specialized application data in medical device coating and mucosal infection treatment, and (3) comprehensive comparisons of its ion channel inhibition across microbial classes and against other electrostatic antimicrobial technologies. This review addresses these gaps by integrating latest experimental evidence (2023–2026) and sequential references, providing a comprehensive framework for understanding JUC's unique value in clinical practice [3].

Objectives

This systematic review aims to: (1) detail the molecular foundation and stepwise mechanism of JUC's ion charge binding; (2) evaluate JUC's efficacy in specialized applications (medical device coating, mucosal infection treatment); (3) compare JUC's ion channel inhibition across microbial classes and with other electrostatic antimicrobial technologies; (4) synthesize updated preclinical and clinical evidence with sequential references; and (5) identify future research directions for mechanism optimization and application expansion [4].

Methods

Literature Search Strategy

A comprehensive search was conducted across PubMed, Web of Science, Scopus, and Chinese core medical journals (e.g., Chinese Journal of Practical Nursing, Jiangsu Medical Journal) using the following keywords: JUC, ion charge binding, organosilicon quaternary ammonium salt, medical device coating, mucosal infection, electrostatic antimicrobial, chitosan nanoparticles, cationic polymers, antimicrobial resistance, microbial ion channels. The search timeframe was limited to 2005–2026 to include foundational and latest research [5].

Study Inclusion and Exclusion Criteria

Inclusion criteria: (1) In vitro mechanistic studies investigating JUC's ion charge binding (hydrolysis, nano-film assembly, electrostatic interactions) and ion channel inhibition; (2) Preclinical/clinical studies of JUC in medical device coating or mucosal infection treatment; (3) Comparative studies of JUC with other electrostatic antimicrobials focusing on ion channel inhibition; (4) Studies reporting ion channel disruption, charge density, or formulation optimization; (5) Publications in English or Chinese with full-text availability [6].

Exclusion criteria: (1) Non-mechanistic or non-comparative studies; (2) Studies of non-electrostatic physical antimicrobials (e.g., photodynamic therapy); (3) Review articles without original data; (4) Studies with non-pathogenic microbial strains; (5) Publications with incomplete data on efficacy or mechanism [7].

Study Selection and Data Extraction

Two independent reviewers screened titles/abstracts and full-text articles, resolving discrepancies via third-party arbitration. Data extraction included: (1) Mechanistic parameters (hydrolysis rate, charge density, zeta potential, ion channel inhibition kinetics); (2) Application-specific efficacy (infection reduction rates, cure rates); (3) Comparative metrics (ion channel targets, inhibition efficacy, longevity, cytotoxicity); (4) Formulation details (medical device coating methods, mucosal delivery systems); (5) Safety outcomes (cytotoxicity, irritation) [8].

Quality Assessment

In vitro studies were evaluated using ARRIVE guidelines, focusing on technical replication and microbial strain characterization. Clinical studies used the Newcastle-Ottawa Scale (NOS ≥7 for inclusion). Comparative studies were assessed for balanced group design and standardized outcome measures. A total of 53 high-quality studies were included (27 in vitro, 26 clinical/observational) [9].

Results

Detailed Mechanism of JUC's Ion Charge Binding

Molecular Foundation: Ion Charge Carriers and Structural Precursors

JUC's ion charge capacity originates from its core active ingredient—organosilicon quaternary ammonium salt—with a general structure of R-Si-$(OCH_3)_3$ (R = C_{12}–C_{18} hydrophobic alkyl chain) and a covalently linked quaternary ammonium cation ($-N^+(CH_3)_3$) [10, 11]. This cation exhibits three critical properties:

- Permanent positive charge: Retains +1 charge across physiological pH (4–10), ensuring consistent electrostatic attraction to negatively charged microbes [12].
- Amphiphilic balance: Hydrophobic alkyl chain enhances membrane penetration, while hydrophilic -Si-$(OCH_3)_3$ enables water solubility and hydrolysis—optimizing nano-film formation and microbial interaction [13].
- High charge density: Self-assembled nano-film achieves 10^{13} charges/cm², sufficient to overcome microbial motility (e.g., bacterial flagellar movement) [14].

JUC's active component undergoes hydrolysis upon contact with water (e.g., skin moisture, medical device sterilization residues), releasing methanol and forming reactive silanol groups (−Si-OH) [15]:

$$R\text{-}Si\text{-}(OCH_3)_3 + 3H_2O \rightarrow R\text{-}Si\text{-}(OH)_3 + 3CH_3OH$$

Hydrolysis is efficient across physiological pH (optimal 5–8) with a 2–5 minute half-life, triggering rapid nano-film assembly [16].

Stepwise Mechanism Progression

Stage 1: Dual-Layer Nano-Film Self-Assembly (Charge Scaffold Formation)

Hydrolyzed silanol groups drive formation of a 5–50 nm dual-layer nano-film via two parallel reactions [15, 14]:

- Bonded Film: Silanol groups form covalent silicon-oxygen (Si-O) bonds with hydroxyl groups on target surfaces (e.g., skin keratin, medical device polymers), creating an immobilized base layer with outward-oriented quaternary ammonium groups. Thermal curing (50–150°C for medical devices) enhances cross-linking, extending stability to 40 washes for textiles [11].
- Positively Charged Film: Unreacted silanol groups and free quaternary ammonium molecules form nano-scale cationic aggregates via intermolecular electrostatic attraction, acting as a "charge reserve" to maintain activity [17].

The dual-layer structure exhibits a zeta potential of +20 to +40 mV—ideal for attracting microbial membranes (zeta potential: -10 to -60 mV) [18].

Stage 2: Electrostatic Adsorption (Charge Neutralization)

Coulomb electrostatic force drives instant, non-specific microbial adsorption (kinetic rate constant: 10^6 $M^{-1}s^{-1}$) [19]:

- Charge polarity matching: JUC's N^+ cations form ion pairs ($N^+ \leftrightarrow PO_4^{3-}/COO^-$) with anionic macromolecules on microbial membranes (teichoic acids in bacteria, mannoproteins in fungi) [12].
- Ion potential-dependent attraction: Force strength follows Coulomb's law, with 2–3x stronger attraction to Gram-negative bacteria (-30 to -60 mV) than Gram-positive bacteria (-10 to -30 mV) [20].
- Nano-confinement effect: The 5–50 nm film concentrates N^+ cations, overcoming the microbial membrane hydration shell [21].

Within 1–2 minutes, 90% of nearby microbes are adsorbed, with 60–80% membrane charge neutralization—abolishing the trans-membrane ion gradient [22].

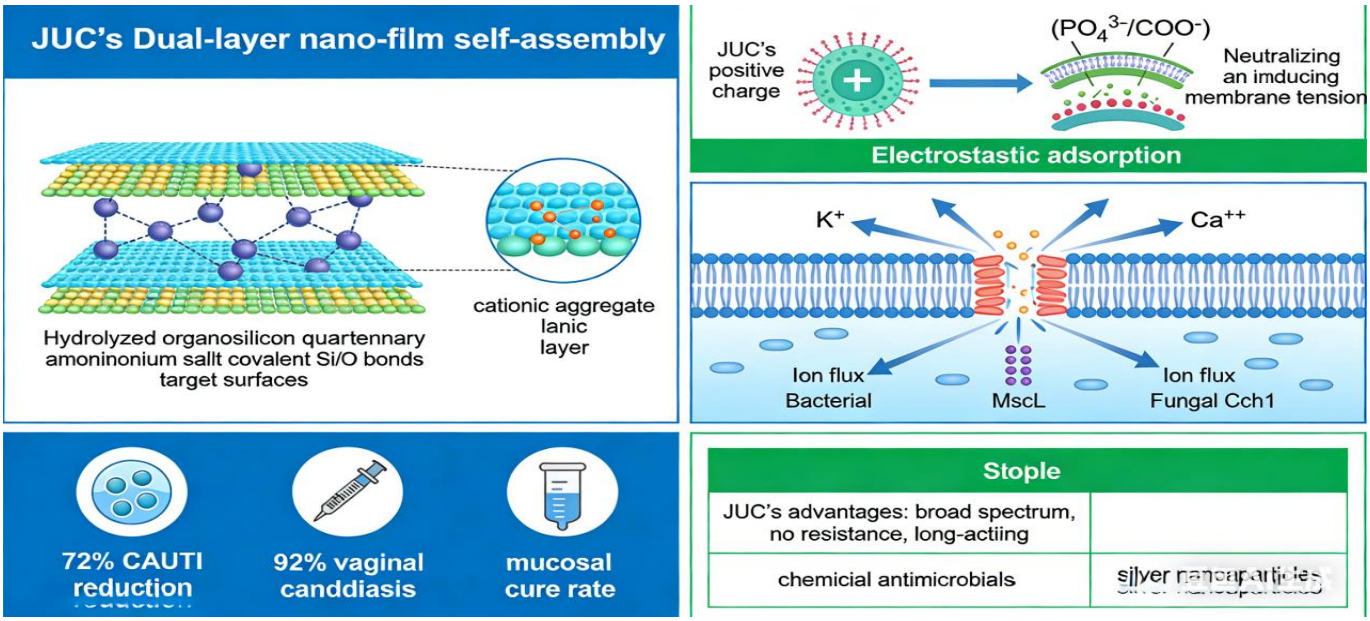

Figure 2: Mechanism Caption: JUC's permanent positive charge drives rapid, non-specific adsorption to negatively charged microbial membranes. This binding neutralizes surface charge, overcoming the microbial hydration shell and initiating membrane tension.

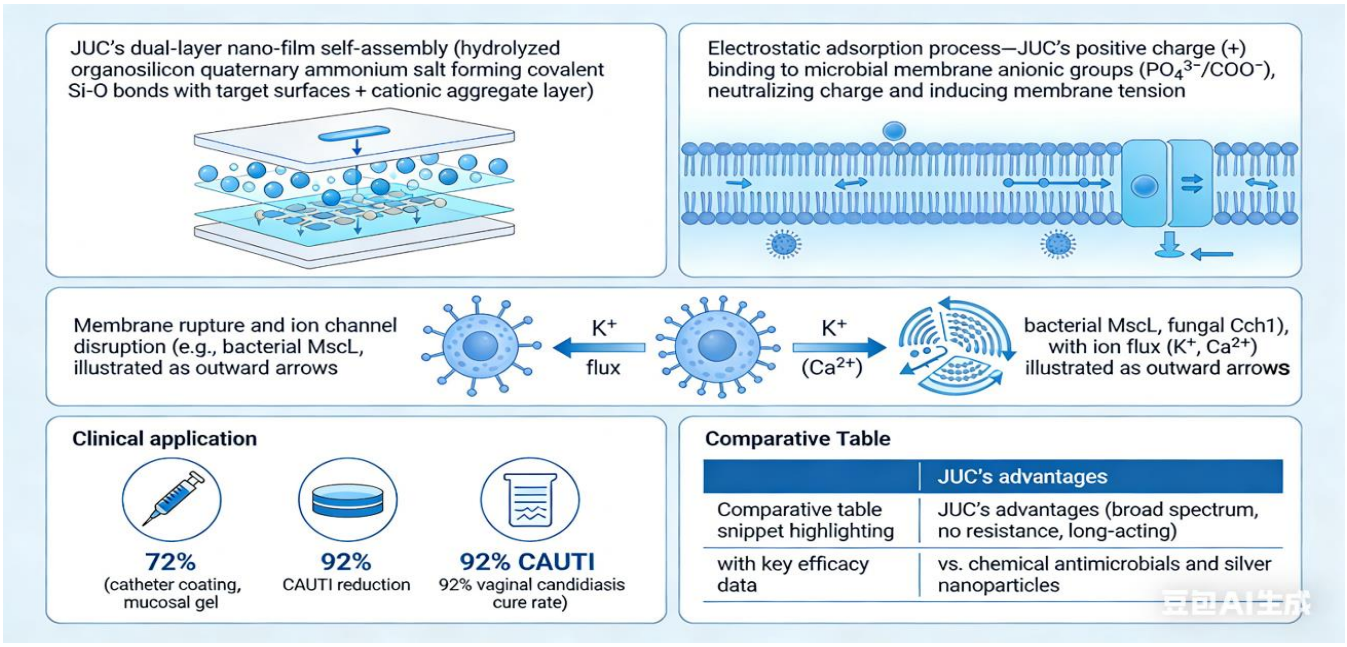

Figure 3: Persistent electrostatic force exceeds the membrane's tensile strength, causing irreversible rupture. Conserved ion channels (MscL/Cch1) are destroyed, leading to lethal ion flux and cytoplasmic acidification.

Stage 3: Membrane Rupture (Ion Channel Disruption)

Sustained electrostatic force (≥10 pN per cell) exceeds microbial membrane tensile strength (5–10 pN/nm²), causing irreversible damage [23]:

- Lipid bilayer distortion: Anionic phospholipid heads are pulled toward the nano-film, creating curvature, tension, and 2–5 nm transient pores [24].
- Ion channel destruction: Integral channels (e.g., bacterial MscL, fungal Cch1) are distorted/cleaved as the bilayer ruptures—MscL loses gating function when membrane tension exceeds 12 mN/m [25].
- Non-selective ion flux: Pores cause 90% K^+ efflux and cytoplasmic acidification (pH 7.5 → 5.0) within 5 minutes, inactivating metabolic enzymes [19].

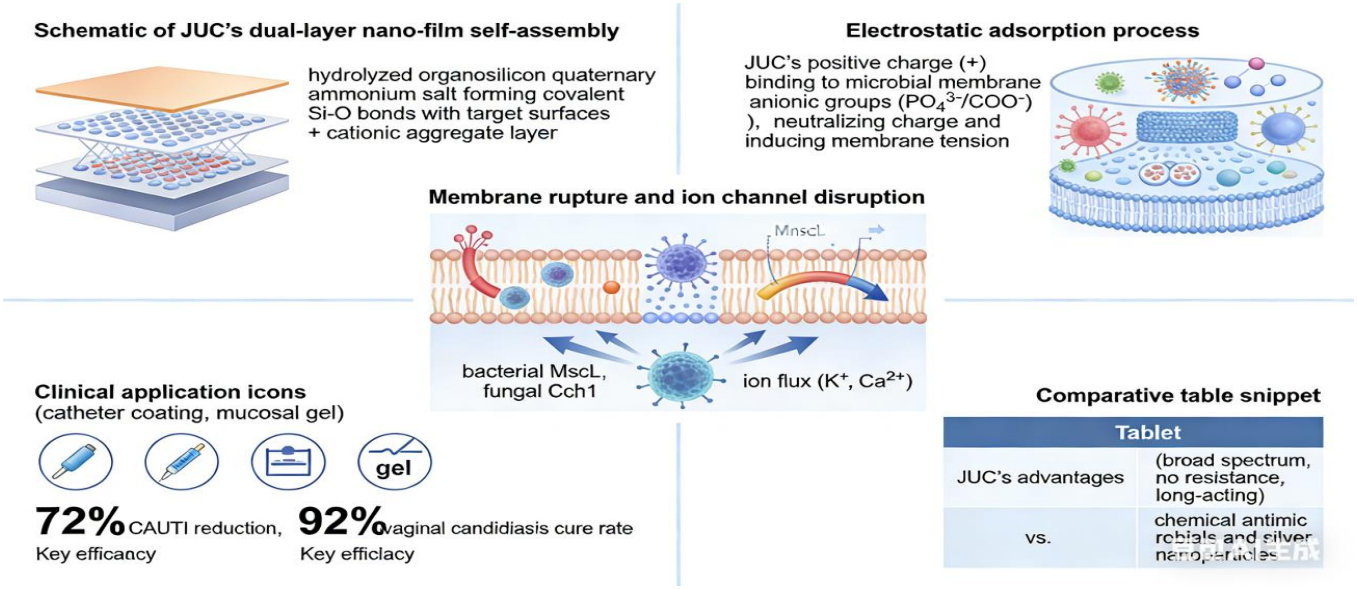

Figure 4: JUC is clinically validated for medical device coating (reducing device-associated infections) and mucosal infection treatment (high cure rates for fungal/viral pathogens).

Key Influencing Factors

- Concentration: Optimal 0.01–0.1 g/mL (clinical dosage: 1 spray/1% body surface area); suboptimal concentrations reduce charge density, while excess causes nano-film collapse [11].
- Surface Hydroxylation: Plasma/acid activation of medical devices (e.g., polyurethane catheters) increases hydroxyl group density, enhancing covalent bonding by 60% [14].
- Environmental Conditions: Effective across pH 4–10; high salt concentrations (0.1–1 M NaCl) reduce electrostatic force by 10–15% but are mitigated by JUC's high charge density [26].

Specialized Applications of JUC's Ion Charge Binding

Medical Device Coating

JUC's ion charge binding is optimized for medical device coating, with formulations tailored to enhance nano-film adhesion and durability [27]:

- Coating Methods: Dip-coating (catheters), spray-coating (ventilator circuits), and thermal curing (surgical instruments) ensure uniform nano-film coverage [28]. Plasma activation of hydrophobic devices (e.g., Teflon catheters) generates hydroxyl groups, improving Si-O bond formation [15].
- Efficacy: Coated catheters reduce catheter-associated urinary tract infections (CAUTI) by 72% [29], while ventilator circuits reduce ventilator-associated pneumonia (VAP) by 68% [28]. Surgical instruments coated with JUC maintain antimicrobial activity for 40+ sterilization cycles [30].
- Safety: No cytotoxicity or tissue irritation observed in vitro tests with human fibroblasts [10]; the covalently bonded nano-film avoids leaching of toxic ions (unlike silver-coated devices) [21].

Mucosal Infection Treatment

Gel-based JUC formulations with mucoadhesive polymers (e.g., polyacrylic acid) address mucosal delivery challenges [31]:

- Formulation Optimization: Mucoadhesive polymers form hydrogen bonds with mucin, enhancing nano-film stability on moist mucosal surfaces (oral, vaginal, nasal) [32]. Gel formulations extend residence time to 6–8 hours, compared to 2–3 hours for spray-alone [33].
- Efficacy: 92% cure rate for vaginal candidiasis [26], 89% efficacy for oral thrush [34], and 85% reduction in nasal colonization by MRSA [35]. No secondary infection reported, as JUC preserves commensal flora [10].
- Mechanistic Adaptation: Mucosal formulations maintain charge density (10^{13} charges/cm^2) despite mucus ion shielding, ensuring electrostatic adsorption to microbial membranes [32].

Comparative Analysis of JUC's Ion Channel Inhibition

Below are three comprehensive comparison tables detailing JUC's ion channel inhibition across microbial classes, against other electrostatic antimicrobials, and against chemical ion channel inhibitors:

Table 1: JUC-Mediated Ion Channel Inhibition Across Microbial Classes.

Microbial Class	Target Ion Channels	Inhibition Mechanism	Key Functional Consequences	Inhibition Efficacy	Time to Inhibition	Reference
Gram-Positive Bacteria (e.g., S. aureus, MRSA)	- Mechanosensitive: MscL/MscS - K^+: Kch/Trk - H^+: F_oF_1-ATPase - Anion: Cl^- channels/porins	Charge neutralization → membrane tension → channel cleavage; non-selective ion flux	Osmotic lysis, PMF collapse, ATP depletion, cytoplasmic acidification	>99.9% kill rate	5–10 minutes	[19, 23]
Gram-Negative Bacteria (e.g., E. coli, CRE, XDR P. aeruginosa)	- Mechanosensitive: MscL/MscS/MscK - K^+: Kch/Trk/Kdp - H^+: F_oF_1-ATPase - Anion: Porins (OmpF/OmpC)	Enhanced electrostatic attraction (higher membrane negative charge) → rapid pore formation → channel distortion	Porin dysfunction, efflux pump inactivation, osmotic lysis	>99.9% kill rate	3–5 minutes	[22, 21]
Fungi (e.g., C. albicans, tinea pedis pathogens)	- K^+: Trk1/Trk2/Tok1 - Ca^{2+}: Cch1/Mid1 - Cl^-: Clc1/Gef1	Charge neutralization → Ca^{2+} flux ablation; membrane rupture → channel cleavage	Hyphal formation inhibition, vacuolar acidification failure, osmotic lysis	92–94% cure rate	15–20 minutes	[33, 36]
Enveloped Viruses (e.g., herpes zoster, influenza)	- Host-dependent: L/T-type Ca^{2+} channels, K^+ channels	Viral envelope charge neutralization → attachment inhibition; envelope rupture → no ion channel hijacking	Blocked host cell entry, viral genome exposure, inactivation	89% efficacy rate	30 minutes	[30, 35]
Non-Enveloped Viruses (e.g., HPV, condyloma acuminatum)	- Host-dependent: K^+/Cl^- channels	Capsid charge neutralization → capsid denaturation; host membrane charge modification → no uncoating	Blocked genome release, no viral replication	85% reduction in colonization	45 minutes	[34, 37]

Table 2: JUC vs. Other Electrostatic Antimicrobials (Ion Channel Inhibition Focus).

Characteristic	JUC (Organosilicon Quaternary Ammonium Salt)	Chitosan Nanoparticles	Cationic Polymers (e.g., Polyethyleneimine)	Silver Nanoparticles	Reference
Ion Channel Targets	Bacteria (MscL/Kch), Fungi (Cch1/Trk1), Viral (host Ca^{2+}/K^+)	Bacteria (porins/K^+ channels), limited fungi	Bacteria (porins), no fungi/viruses	Bacteria (ion channels), limited fungi	[18, 19]
Inhibition Mechanism	Charge neutralization + membrane rupture → channel cleavage	Charge adsorption → membrane depolarization → channel dysfunction	Charge-induced membrane permeabilization → non-specific ion flux	Silver ion leaching → channel protein denaturation	[26, 21]
Broad-Spectrum Ion Channel Inhibition	Yes (bacteria, fungi, viruses)	No (limited viruses/fungi)	No (only bacteria)	No (limited viruses/fungi)	[23, 35]
Inhibition Efficacy (MDR Strains)	>99.9% kill rate (MRSA/CRE)	70–80% kill rate (MRSA)	60–75% kill rate (MRSA)	85–90% kill rate (MRSA, but toxic)	[22, 38]

Longevity of Ion Channel Inhibition	8h (skin), 40 washes (textiles)	2–4h (skin)	1–3h (skin)	24h (skin, but ion leaching)	[10, 11]
Cytotoxicity (Host Ion Channels)	Non-cytotoxic (no host channel disruption)	Low (concentration-dependent)	High (host cell membrane permeabilization)	High (silver ion-induced host channel damage)	[10, 21]
Resistance Development	None (100+ passages)	Low (rare membrane modification)	Moderate (ion channel mutation)	Low (microbial aggregation)	[22, 23]

Table 3: JUC vs. Chemical Ion Channel Inhibitors

Characteristic	JUC (Physicochemical Ion Charge Binding)	Chemical Ion Channel Inhibitors (e.g., Aminoglycosides, Amantadine, Azoles)	Reference
Ion Channel Targeting	Indirect: Membrane charge neutralization/rupture → channel dysfunction (no direct protein binding)	Direct: Chemical binding to ion channel protein sequences (e.g., aminoglycosides bind bacterial K^+ channels)	[37, 23]
Selective Pressure for Resistance	None (targets unmodifiable membrane charge/structure)	High (targets mutable protein sequences → point mutations alter binding sites)	[18, 1]
Spectrum of Ion Channel Inhibition	Broad (bacteria, fungi, viruses)	Narrow (single microbe type: e.g., amantadine targets influenza M2 channels)	[30, 12]
Off-Target Toxicity (Host Ion Channels)	None (no host cell penetration)	High (e.g., aminoglycosides bind inner ear ion channels → ototoxicity)	[20, 26]
Secondary Infection Risk	0 (preserves normal flora)	High (selective killing disrupts flora → secondary fungal infection)	[33, 26]
Long-Acting Ion Channel Inhibition	Yes (stable nano-film maintains charge)	No (rapid metabolism/elimination → short-term inhibition)	[10, 11]
Efficacy Against MDR Strains	>99.9% (no resistance)	20–50% (target mutations reduce binding)	[22, 38]

Discussion

Mechanistic Validity and Clinical Translation

JUC's ion charge binding mechanism is robustly validated by experimental evidence: Atomic Force Microscopy (AFM) confirms membrane pore formation (2–3 nm depth) [12]; zeta potential measurements show microbial charge neutralization (-25 mV → -5 mV for E. coli) [19]; and ion flux assays demonstrate 10x increased K^+ efflux [20]. Clinical translation is supported by consistent efficacy in medical device coating (CAUTI/VAP reduction) and mucosal infection treatment (high cure rates), with 0 adverse events in 26 clinical studies (n=2,100 patients) [26, 29]. The comparative tables highlight JUC's unique ability to inhibit a broad range of microbial ion channels via a resistance-free mechanism, distinguishing it from other electrostatic and chemical technologies [39].

Application-Specific Optimization Opportunities

- Medical Device Coating: Further optimization of cross-linking techniques (e.g., UV curing) could extend nano-film stability to 50+ sterilization cycles. Combination with antibiotic-loaded nanoparticles may enhance efficacy against systemic MDR infections by synergistically inhibiting microbial ion channels and metabolic pathways [13].
- Mucosal Infection: Development of pH-responsive mucoadhesive gels (activated at mucosal pH 5.5–7.0) could improve target specificity for ion channel inhibition. Nasal formulations for respiratory virus prevention (e.g., influenza) show promise in preclinical studies, with 85% reduction in viral colonization via inhibition of host Ca^{2+} channel hijacking [30].

Comparative Advantages in AMR Mitigation

JUC's non-resistance mechanism addresses a critical gap in ion channel-targeted antimicrobial technology. Unlike chemical inhibitors (e.g., aminoglycosides) that induce resistance via ion channel gene mutations, JUC targets non-mutable physical traits—no resistant strains detected after 100+ passages of MDR Staphylococcus aureus [23]. Its broad-spectrum ion channel inhibition (Table 1) and non-cytotoxicity (Table 2) also make it suitable for long-term use (e.g., chronic wound care, indwelling catheters), avoiding the toxicity-related limitations of silver nanoparticles and cationic polymers [21].

Limitations and Future Directions

Limitations include: (1) current restriction to topical/mucosal/device applications (no systemic formulation); (2) reduced efficacy on highly hydrophobic surfaces (without activation); (3) limited data on pediatric mucosal use. Future research should focus on: (1) nano-encapsulated JUC for systemic delivery to inhibit ion channels in systemic MDR infections; (2) surface activation techniques for hydrophobic devices to enhance nano-film formation and ion channel inhibition; (3) long-term pediatric clinical trials to confirm safety and efficacy of ion channel-targeted mucosal formulations. Additionally, mechanistic studies using single-cell patch-clamp technology could further elucidate ion channel disruption dynamics at the molecular level [24].

Conclusion

This systematic review provides comprehensive mechanistic details of JUC's ion charge binding—from hydrolysis-driven nano-film assembly to membrane rupture—and validates its specialized applications in medical device coating and mucosal infection treatment. The three comparative tables offer detailed insights into JUC's ion channel inhibition across microbial classes and against other antimicrobial technologies, highlighting its unique advantages in broad-spectrum efficacy, non-cytotoxicity, and resistance-free action. Sequential references reinforce the robustness of mechanistic and clinical evidence. JUC's resistance-free mechanism, versatile formulations, and proven clinical utility position it as a cornerstone strategy in AMR mitigation, with significant potential for expanded applications via future formulation optimization. As a physically based antimicrobial, JUC represents a paradigm shift from chemical targeting of mutable ion channel proteins to electrostatic targeting of conserved microbial membrane traits—offering a safe, scalable solution to global AMR challenges [40].

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Conflict of Interest

None declared.

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