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ORIGINAL RESEARCH

Anti-Virulence Efficacy of Chinese Dragon's Blood Against *Pseudomonas aeruginosa* Isolated from Wound Infections

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Background: *Pseudomonas aeruginosa* (*P. aeruginosa*) wound infections are an emerging global health threat. Empiric therapy of infected wounds with Chinese dragon's blood (CDB) is one of the most precious traditional Chinese medicines used in clinical settings. We investigated the anti-virulence efficacy of CDB against *P. aeruginosa* isolated from wound infections.

Methods: We collected six *P. aeruginosa* clinical isolates obtained from wound specimens. Antimicrobial susceptibility profiles were determined using the agar dilution method. Biofilm formation and eradication assays, quantitative real-time PCR (RT-qPCR), and bacterial motility assays were performed to evaluate the efficacy of CDB on biofilm formation, mature biofilm eradication, and motility ability of *P. aeruginosa* isolates.

Results: Minimal inhibitory concentration (MIC) values of CDB against *P. aeruginosa* isolates were ≥1024 μg/mL. The differences in biofilm formation ability between the CDB-containing LB broth and LB broth groups were statistically significant ($P < 0.05$). The results of mature biofilm-eradicating assays indicated that CDB had excellent efficacy on eradicating the biofilm formed by all experimental strains ($P < 0.05$). The mRNA relative expression of *lasR, pslA, pelA, algD, and algU* genes in *P. aeruginosa* strains was significantly downregulated after exposure to CDB at a concentration of 128 μg/mL ($P < 0.05$). CDB could inhibit the motility ability of *P. aeruginosa* isolates through swimming, swarming, and twitching motilities.

Conclusion: CDB exerts a positive anti-virulence efficacy on *P. aeruginosa*. CDB significantly reduced the biofilm formation by downregulating the mRNA relative expression of the biofilm-associated genes *lasR, pslA, pelA, algD, and algU*. In addition, CDB efficiently inhibited the motility ability of *P. aeruginosa* isolates by swimming, swarming, and twitching motilities in a concentration-dependent manner. Therefore, these findings position CDB as an alternative for *P. aeruginosa* wound infections management in clinical settings.

Keywords: Chinese dragon's blood, wound infections, *Pseudomonas aeruginosa*, anti-virulence efficacy

Introduction

Wound infections are emerging as a major global health challenge, and studies have demonstrated that approximately 52–70% of trauma patients succumb to wound infections.[1,2] While acute wounds generally exhibit self-healing capacity without requiring substantial clinical intervention, chronic wounds frequently fail to undergo spontaneous repair, which necessitates therapeutic management.[3] Clinically, the treatment of chronically infected wounds poses significant difficulties owing to microbial colonization of the wound beds. This pathological colonization disrupts the physiological healing cascade, leading to impaired tissue regeneration and, ultimately, non-healing wound states.[4,5] Notably, *Pseudomonas aeruginosa* (*P. aeruginosa*) and *Staphylococcus aureus* (*S. aureus*) are the most common pathogenic

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microorganisms isolated from infected wounds, and their ability to express virulence determinants and establish biofilm structures may adversely affect the wound healing process.[6] Biofilms constitute structured microbial communities encased in a self-synthesized matrix composed of extracellular polymeric substances (EPS) derived from microbial and host sources.[7,8] These biofilm-forming bacteria demonstrate distinct metabolic profiles embedded within this protective macromolecular architecture. Biofilm facilitates environmental adaptation through multifaceted mechanisms, thereby conferring intrinsic tolerance to conventional antimicrobial interventions via physical exclusion and altered microbial physiology.[9,10] Some studies have revealed that quorum sensing system contributes to biofilm formation in *P. aeruginosa* wound isolates. Therefore, biofilm formation by *P. aeruginosa* critically contributes to the pathogenesis of non-healing wound infections. In addition, the incorporation of antibiotics into therapeutic regimens has proven to be effective in eradicating diverse pathogens. However, the widespread and often indiscriminate use of broad-spectrum antimicrobial agents has driven the progressive emergence of antimicrobial resistance (AMR).[11,12] This escalating prevalence of AMR has precipitated formidable challenges in clinical infection management, compounding the therapeutic complexities of combating persistent infections.[4]

Within the framework of integrated medical approaches, traditional Chinese medicine (TCM) formulations have emerged as viable adjunctive treatment modalities for wound infection management, with Chinese dragon's blood (CDB) being a particularly notable botanical agent.[13,14] Pharmacopeial specifications (National Drug Standard WS_3-082 (Z-016)-99(Z)) define CDB as a crimson-hued resinous exudate derived from the ligneous tissues of *Dracaena cochinchinensis* (*Liliaceae* family).[15] There are some studies identify proanthocyanidins as the predominant phytochemical group in terms of relative quantity. Additionally, other significant phytochemical groups include alkaloids, diterpenoids, phytosteroids, saponins, phenolics, and polyphenolics.[16,17] The pharmacological studies have demonstrated that this phytotherapeutic substance contains multiple bioactive compounds with anti-inflammatory, antimicrobial, antifungal, and cytotoxic properties, rendering it applicable to diverse pathological conditions.[18] Results from an in vivo study on burn wounds showed that scaffolds containing 20% CDB exhibited excellent wound healing ability with 80.3% wound closure after 21 days,[19] and the novel research illustrated the potential protective mechanism of CDB on intestinal inflammatory-related diseases and might be useful for further clinical application of CDB.[20] Particularly relevant to wound care, CDB has been employed therapeutically in clinical practice to address suppurative lesions, diabetic foot complications, and traumatic tissue damage, among other conditions.[14] The present investigation was designed to systematically evaluate the capacity of CDB to attenuate virulence determinants in *P. aeruginosa* isolates, while concurrently establishing preclinical evidence to optimize its clinical application in wound infection management.

Materials and Methods

Bacterial Isolates and Growth Conditions

Six *P. aeruginosa* clinical isolates were obtained from wound specimens of patients receiving treatment at the First Affiliated Hospital of Huzhou University (Zhejiang Province, China) in 2024. Bacterial identification was confirmed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) using the VITEK MS platform (bioMérieux, Lyon, France). The microbial cultures were cryopreserved at −80°C and subsequently subcultured on blood agar medium under standardized conditions (37°C for 18–24 h) prior to experimental analysis.

Determination of Antimicrobial Resistance Profiles

Antimicrobial susceptibility profiles were determined using the agar dilution technique in compliance with the latest Clinical and Laboratory Standards Institute guidelines (CLSI 2025).[21] The experimental protocol involved resuspending overnight cultures in sterile saline solution (0.85% NaCl), with subsequent turbidity standardization to match the 0.5 McFarland reference (approximately 1.5 × 10□ CFU/mL). Ten-fold dilutions of the bacterial suspensions were then inoculated onto Mueller–Hinton agar plates containing antimicrobial agents. Following 16–18 h of aerobic incubation at 37°C, the inhibition zones were systematically evaluated. The investigational compound CDB (Lot Z25O9B84376, Yuanye Biotechnology, Shanghai, China) was dissolved in dimethyl sulfoxide to create serial dilutions spanning 1–512 μg/mL. Strain validation was performed using *P. aeruginosa* ATCC 27853 as the reference organism. Minimal

inhibitory concentration (MIC) determinants were conducted through independent experiments in triplicate to ensure methodological reproducibility.

Biofilm Formation and Eradication Assays

Biofilm formation and eradication assays were performed according to the methods described by Bukhari, with some modifications.[22,23] This study was carried out using seven *P. aeruginosa* strains (PA676, PA679, PA731, PA732, PA764, PA769, and PAO1). A single bacterial colony isolated from the blood agar plate was inoculated into 3 mL fresh LB medium and incubated overnight at 37°C with continuous agitation (180 rpm). The bacterial suspension was standardized to 0.5 McFarland units using sterile normal saline, followed by a 1:100 dilution in fresh LB broth. Aliquots (100 μL) were transferred to 96-well microplates containing different concentrations of CDB (0, 32, 64, 128, or 256 μg/mL). Following 24-hour incubation at 37°C, non-adherent cells were removed through two consecutive washes with 200 μL of phosphate-buffered saline (PBS, 1×, Sigma-Aldrich). The microplates were air-dried and inverted at ambient temperature. Subsequent staining was performed using 150 μL of 1% (*w*/*v*) crystal violet (CV; Solarbio Biotechnology, Beijing, China; Lot number: 20240846) for 15 min at room temperature. Excess stain was discarded and the wells were rinsed three times with PBS. Bound CV was eluted with 150 μL of destaining solution (95% ethanol: 5% glacial acetic acid, *v*/*v*), and biofilm quantification was achieved by measuring the optical density at 595 nm using a Multiskan FC microplate reader. All experimental conditions were tested in triplicate with three independent biological replicates. Biofilm formation and mature biofilm eradication experiments differed according to the timing of the drug addition. The drug was added before the biofilm formed in the biofilm formation experiment and after the biofilm had matured in the eradication experiments.

Quantitative Real-Time PCR (RT-qPCR)

All *P. aeruginosa* strains (PA676, PA679, PA731, PA732, PA764, PA769, and PAO1) were either exposed to CDB at a concentration of 128 μg/mL or left untreated. Total RNA was subsequently isolated from bacterial cultures using the Bacterial RNA Miniprep Kit (Biomiga, Shanghai, China). First-strand cDNA synthesis was performed with 1000 ng of RNA template per reaction using the RevertAid First Strand cDNA Synthesis System (Thermo Scientific, Waltham, MA, USA), following the manufacturer's recommended protocols. Gene mRNA relative expression analysis was conducted via quantitative real-time PCR (RT-qPCR) targeting *lasR, pslA, pelA, algD*, and *algU* transcripts. The *rpsL* housekeeping gene served as an endogenous control for normalization. Gene-specific primers (listed in Table 1) were employed for amplification using SYBR Green chemistry with TB Green Premix Ex Taq II (Tli RNaseH Plus; Takara, Japan) on a QuantStudio 6 Flex Real-Time PCR System (Applied Biosystems, USA).[24] Thermal cycling conditions consisted of initial denaturation at 95°C for 30s, followed by 40 cycles at 95°C for 5s and 60°C for 34s, with melt curve analysis for reaction specificity verification.

Table 1 Primers Used for RT-qPCR in This Study

Gene	Primer	Sequence (5′→3′)	Annealing Temperature
rpsL	Forward	GTGGTGAAGGTCACAACCTG	55°C
	Reverse	CCTGCTTACGGTCTTTGACA	
lasR	Forward	ACGCTCAAGTGGAAAATTGG	58°C
	Reverse	TCGTAGTCCTGGCTGTCCTT	
pslA	Forward	GGCCTGTTTCCCTACCT	55°C
	Reverse	GCGGATGTCGTGGTTG	
pelA	Forward	GGCCTGCTCGAATACCTC	58°C
	Reverse	TGACCTTGAGTTTCTGCGACA	
algD	Forward	CGAGAAGTCCGAACGCCACAC	60°C
	Reverse	ATCGGCGGGAAGTCGTA	
algU	Forward	GATTGATCGTGCGGTTCGTG	58°C
	Reverse	AAGATCCGCGACCGTACCGT	

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Bacterial Motility Assays

Microbial motility assays were conducted using standardized agar-based platforms, fabricated according to established protocols.[25,26] For swarming and swimming behavior assays, 2 μL aliquots of overnight bacterial cultures were centrally inoculated onto low-viscosity (0.3% agar) and high-viscosity (0.5% agar with 0.5% glucose) substrates, respectively. Twitching motility assay involved vertical inoculation of bacterial suspensions at the LB plate (1% agar) interface through stab inoculation. Following 24-hour aerobic incubation at 37°C, radial migration distances from the inoculation site were systematically quantified using digital caliper measurements. For twitching motility assay, removed the agar and subsequent staining was performed using 1 mL of 1% (*w*/*v*) crystal violet for 15 min at room temperature. Then, excess stain was discarded and the wells were rinsed three times with PBS. All experiments were performed in triplicate to ensure methodological reproducibility.

Statistical Analysis

All experimental procedures were performed in triplicate with independent biological repetitions. Quantitative data are presented as mean values ± standard deviation (SD), and intergroup comparisons were analyzed using the one-way ANOVA. Statistical computations were performed using GraphPad Prism, version 9.02 (GraphPad Software Inc., San Diego, CA, USA), with two-tailed analyses employed throughout the study. Statistical significance was set at $P < 0.05$.

Results

Determination of MICs of CDB and Antimicrobial Agents Against *P. aeruginosa* Isolates

Antimicrobial susceptibility testing using the agar dilution method revealed that the MIC values of CDB against *P. aeruginosa* isolates were ≥1024 μg/mL (Table 2).

Moreover, three *P. aeruginosa* isolates (PA676, PA764, and PA767) were resistant to routine clinical antimicrobial agents, including piperacillin/tazobactam (TZP), ceftazidime (CAZ), cefepime (FEP), imipenem (IPM), meropenem (MEM), ciprofloxacin (CIP), and levofloxacin (LVX) (Table 2).

Efficacy of CDB on Biofilm Formation and Mature Biofilm Eradication of *P. aeruginosa* Isolates

The effects of CDB on biofilm formation and mature biofilm eradication of *P. aeruginosa* isolates were investigated using crystal violet staining. Our results illustrated the biofilm structures of *P. aeruginosa* isolates in LB broth in the presence and absence of CDB and noted that the differences in biofilm formation ability between the drug-containing LB broth and LB broth groups were statistically significant ($P < 0.05$; Figure 1). Figure 2 shows the results of the mature biofilm-

Table 2 Minimum Inhibitory Concentrations (MICs) of CDB and Clinical Routine Antimicrobial Agents Against Six *P. aeruginosa* Isolates

Isolates	CDB MICs (μg/mL)	Antibiotic MICs (μg/mL)									
		TZP	CAZ	FEP	IPM	MEM	CIP	LVX	GEN	TOB	AMK
PA676	≥1024	≥128/4^{R}	≥64^{R}	32^{R}	≥16^{R}	8^{R}	2^{R}	4^{R}	2	≤1	4
PA679	≥1024	≤4/4	4	≤1	2	1	≤0.25	0.5	≤1	≤1	≤2
PA731	≥1024	≤4/4	2	2	2	1	≤0.25	0.5	≤1	≤1	≤2
PA732	≥1024	≤4/4	≤1	≤1	2	1	≤0.25	≤0.25	≤1	≤1	≤2
PA764	≥1024	≥128/4^{R}	≥64^{R}	≥64^{R}	≥16^{R}	4	2^{R}	4^{R}	4	≤1	4
PA767	≥1024	64/4	≥64^{R}	16	≥16^{R}	4	2^{R}	2	2	≤1	4

Abbreviations: MICs, minimum inhibitory concentrations; CDB, Chinese dragon's blood; TZP, piperacillin/tazobactam; CAZ, ceftazidime; FEP, cefepime; IPM, imipenem; MEM, meropenem; CIP, ciprofloxacin; LVX, levofloxacin; GEN, gentamicin; TOB, tobramycin; AMK, amikacin;^{R}, resistance.

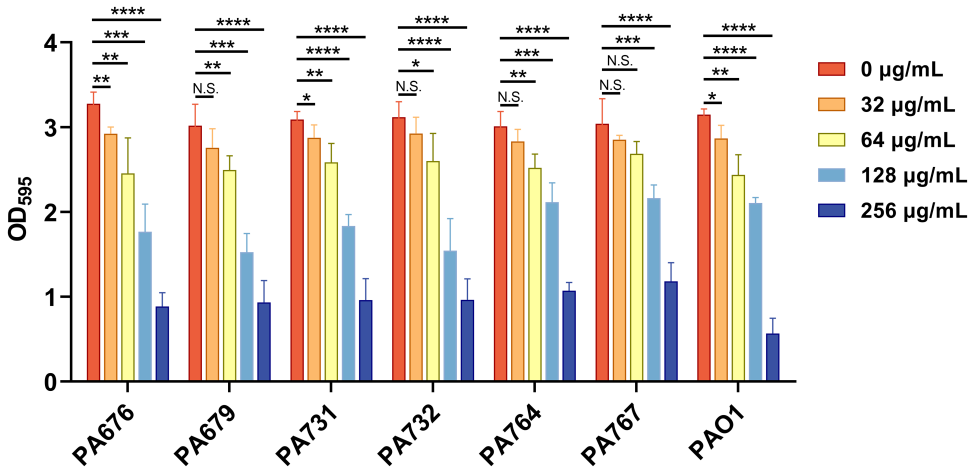

Figure 1 Effects of different concentrations of CDB on the biofilm formation ability of *P. aeruginosa* strains. N.S., $P > 0.05$; *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; ****$P < 0.0001$.

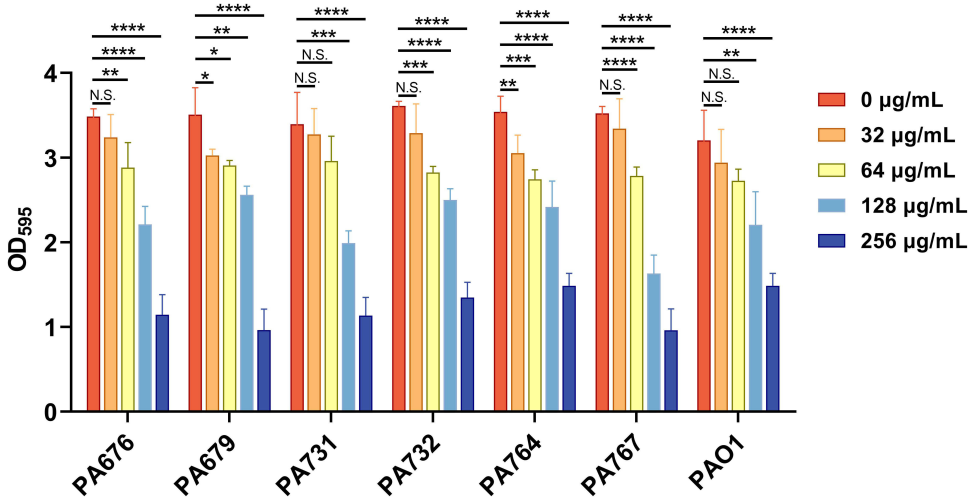

Figure 2 Effects of different concentrations of CDB on the mature biofilm eradication of *P. aeruginosa* strains. N.S., $P > 0.05$; *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; ****$P < 0.0001$.

eradicating assays, which indicated that CDB had excellent efficacy on eradicating the biofilm formed by all experimental strains ($P < 0.05$).

Expression of Genes Involved in Biofilm Formation of *P. aeruginosa* Strains

RT-qPCR results revealed that mRNA relative expression levels of *lasR, pslA, pelA, algD*, and *algU* genes in *P. aeruginosa* strains were significantly downregulated after exposure to CDB at a concentration of 128 μg/mL ($P < 0.05$; Figure 3A–G). These results indicated that CDB could decrease the capacity of biofilm formation of *P. aeruginosa* strains through the inhibition of the mRNA relative expression of *lasR, pslA, pelA, algD*, and *algU* genes.

Efficacy of CDB on the Motility of *P. aeruginosa* Isolates

To study the effect of CDB on the motility of *P. aeruginosa* isolates, motility assays were carried out. By measuring the diffusion diameter of *P. aeruginosa* on the surface of LB plate, the effects of CDB on the mobility of *P. aeruginosa* was analyzed. As shown in Figures 4–6, we concluded that CDB could inhibit the motility ability of *P. aeruginosa* isolates through swimming, swarming, and twitching motilities in a concentration-dependent manner.

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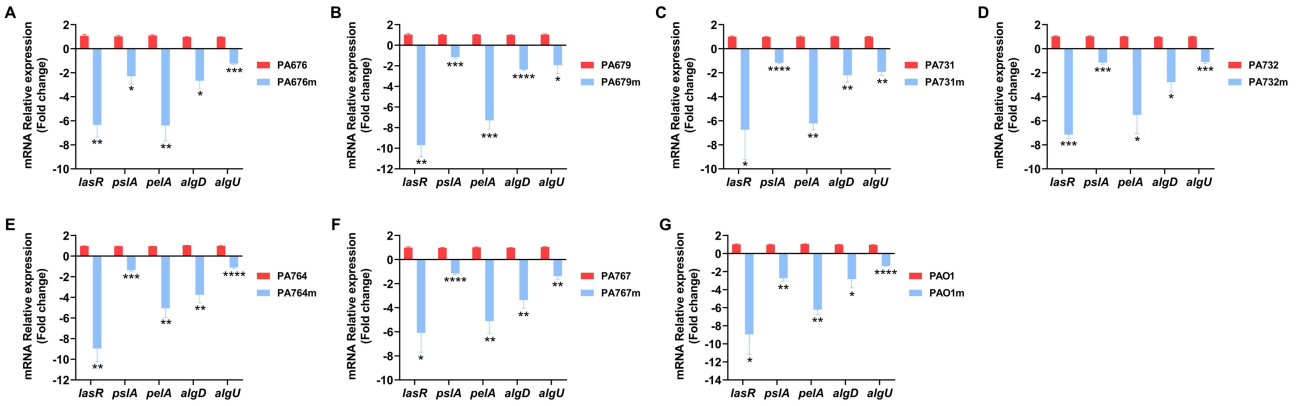

Figure 3 mRNA relative expression levels of *lasR, psIA, pelA, algD, and algU* genes in parental *P. aeruginosa* and corresponding CDB-treated *P. aeruginosa* strains after the exposure of CDB at a concentration of 128 μg/mL. (**A**) *P. aeruginosa* PA676 and PA676m; (**B**) *P. aeruginosa* PA679 and PA679m; (**C**) *P. aeruginosa* PA731 and PA731m, (**D**) *P. aeruginosa* PA732 and PA732m, (**E**) *P. aeruginosa* PA764 and PA764m, (**F**) *P. aeruginosa* PA767 and PA767m, (**G**) *P. aeruginosa* PAO1 and PAO1m; m indicates CDB-treated *P. aeruginosa* strains; **P* < 0.05; ***P* < 0.01; ****P* < 0.001; *****P* < 0.0001.

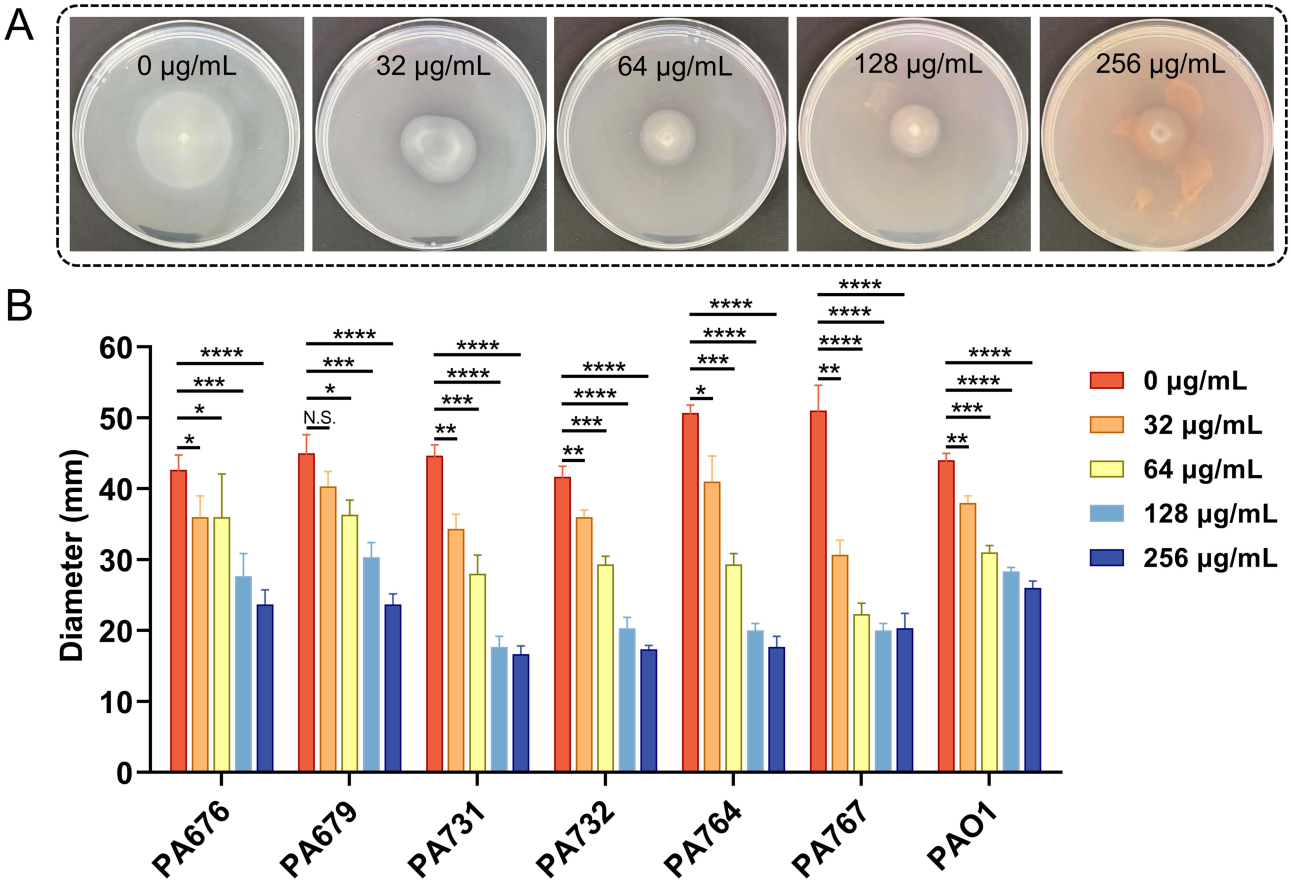

Figure 4 Effects of different concentrations of CDB on the swimming motility ability of *P. aeruginosa* strains. (**A**) Swimming motility ability of *P. aeruginosa* PA767 isolate; (**B**) Swimming motility ability of all *P. aeruginosa* strains; N.S., *P* > 0.05; **P* < 0.05; ***P* < 0.01; ****P* < 0.001; *****P* < 0.0001.

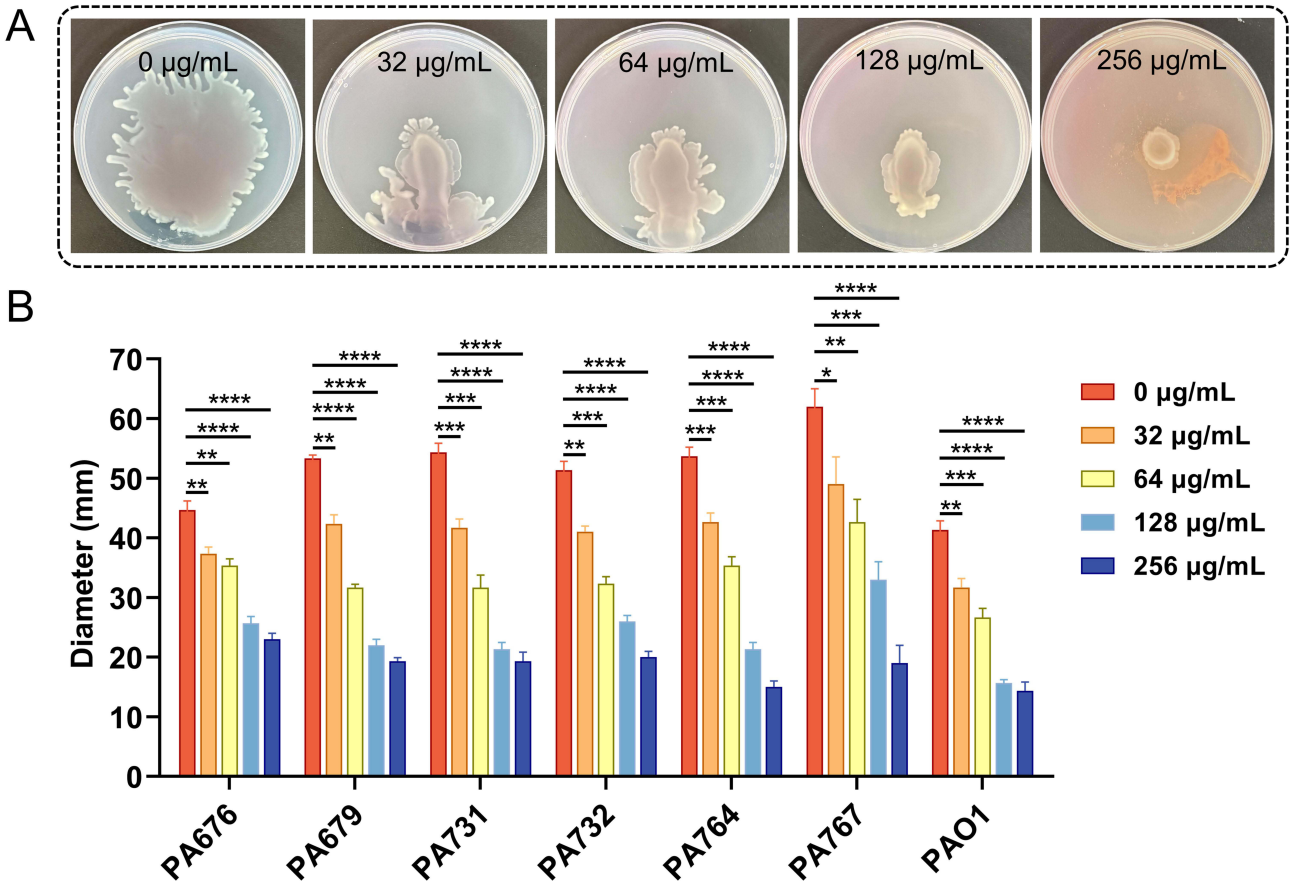

Figure 5 Effects of different concentrations of CDB on the swarming motility ability of *P. aeruginosa* strains. (**A**) Swarming motility ability of *P. aeruginosa* PA767 isolate; (**B**) Swarming motility ability of all *P. aeruginosa* strains; **P* < 0.05; ***P* < 0.01; ****P* < 0.001; *****P* < 0.0001.

Discussion

Clinically, infected wounds not only impair regenerative cascades but also precipitate life-threatening complications through systemic dissemination.[4] Empirical data have identified *P. aeruginosa* and *S. aureus*, particularly multidrug-resistant strains, as the predominant etiological agents in refractory wound infections.[1] Emerging evidence highlights CDB as a promising phytotherapeutic agent, demonstrating quorum-sensing attenuation and biofilm disruption capacities.[14,18] However, CDB is currently used empirically in clinical practice for wound infection treatment, and there is no standardized dosage used in clinical practice in China. Our investigation delineates the mechanistic basis of the anti-pathogenic efficacy of CDB against *P. aeruginosa* clinical isolates obtained from wound specimens, providing preclinical validation for integrating CDB-derived compounds into combinatorial antimicrobial strategies targeting wound infection recalcitrance.

Our previous studies have documented how CDB exerts positive antibacterial efficacy against *S. aureus* and could also reduce biofilm formation and retard the virulence factor alpha-hemolysin of *S. aureus* by downregulating the expression levels of *saeR, saeS*, and *hla* genes.[27] As a clinically comparable pathogen causing wound infections, *P. aeruginosa* also deserves further parallel investigation. Based on the results of the antimicrobial susceptibility testing, we found that CDB exhibits no significant antibacterial activity against *P. aeruginosa* at a concentration of 1024 μg/mL. Fortunately, CDB showed positive anti-virulence efficacy against *P. aeruginosa* isolated from wound infections. Sub-inhibitory concentrations of CDB could reduce the biofilm formation ability of *P. aeruginosa*, and CDB showed excellent efficacy on eradicating the biofilm formed by all experimental strains. These findings were further supported by the RT-qPCR results. We further found that CDB could downregulate the mRNA relative expression of *lasR, pslA, pelA, algD*, and *algU* genes and inhibit the motility of *P. aeruginosa* isolates.

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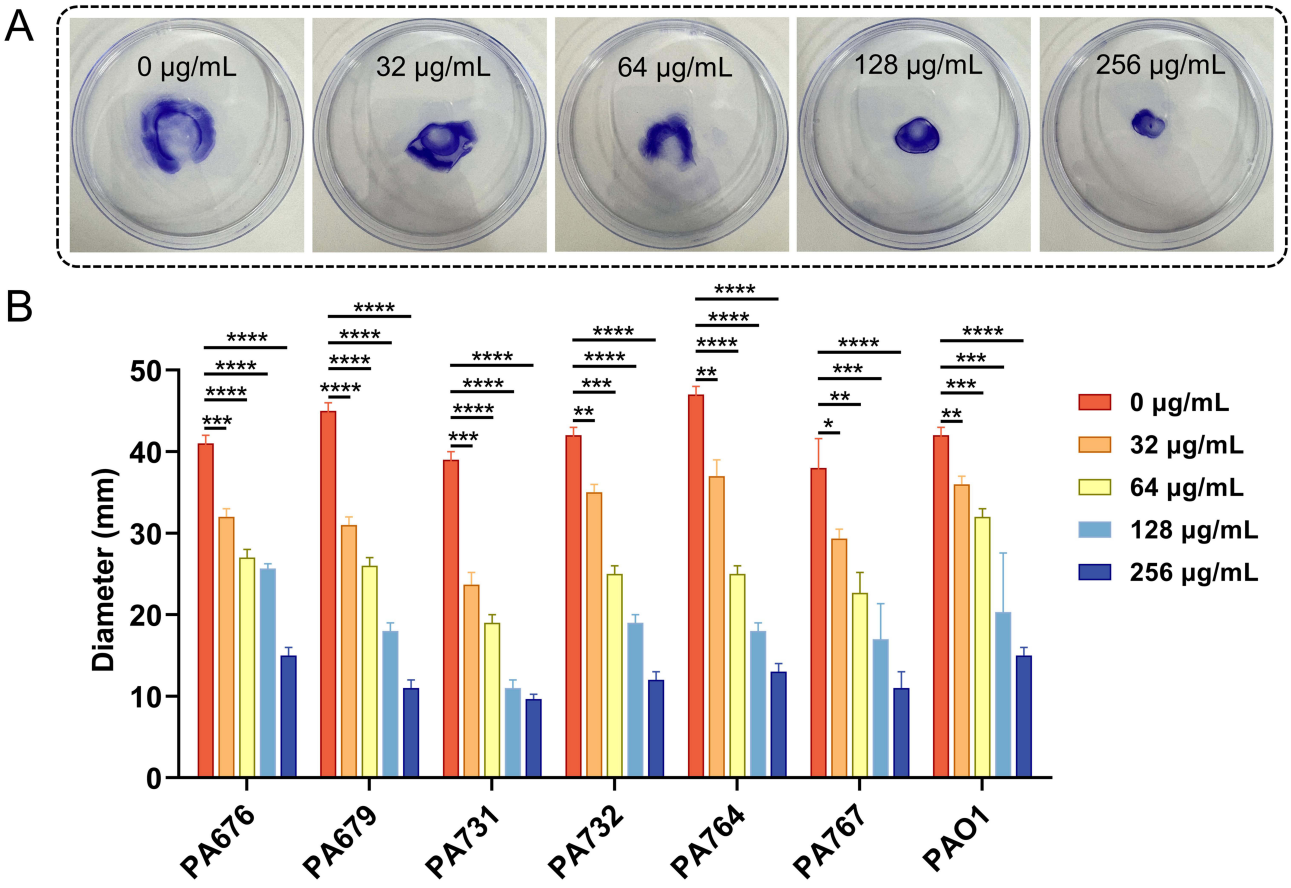

Figure 6 Effects of different concentrations of CDB on the twitching motility ability of *P. aeruginosa* strains. (**A**) Twitching motility ability of *P. aeruginosa* PA767 isolate; (**B**) Twitching motility ability of all *P. aeruginosa* strains; **P* < 0.05; ***P* < 0.01; ****P* < 0.001; *****P* < 0.0001.

Previous investigations have established that, within the microenvironment of chronically infected wounds, bacterial biofilm formation plays a pivotal role in microbial pathogenesis. Biofilms enhance microbial adhesion to wound beds, evade host immune defenses, and promote environmental adaptability.[28–30] Consequently, the development of novel therapeutic compounds targeting biofilm formation has emerged as a critical imperative in wound management research. Experimental protocols for biofilm formation and mature biofilm eradication are distinguished by the temporal administration of therapeutic agents.[22,23] In our study, biofilm formation and eradication assays revealed that sub-inhibitory concentrations of CDB could effectively reduce the biofilm formation ability and eradicating the mature biofilm formed by all experimental *P. aeruginosa* strains. *P. aeruginosa* biofilm formation is a critical determinant of wound healing trajectory and patient mortality in wound infections. Complementing this pathogenic mechanism, Ho et al demonstrated that CDB stimulates angiogenesis, while enhancing keratinocyte proliferation (1.8-fold) and fibroblast migration rates (42% increase vs control).[31] We hypothesized that the dual modulatory effects of CDB, which simultaneously counteract biofilm-mediated healing impairment and activate tissue regenerative pathways, collectively contribute to its therapeutic efficacy in recalcitrant wound management.

Moreover, RT-qPCR was performed to detect the expression of genes involved in biofilm formation of *P. aeruginosa* strains. Interestingly, we found that the mRNA relative expression levels of *lasR* gene in *P. aeruginosa* strains were significantly downregulated after exposure to CDB at a concentration of 128 μg/mL. The quorum sensing (QS) system enables bacterial consortia to synchronize virulence factor secretion, biofilm maturation, metabolic reprogramming, and antimicrobial resistance phenotypes.[32] Previous studies have shown that the *lasRI* gene in the *las* system is upstream of other subsystems and is the main control factor of the quorum sensing system network, and the expression of other

subsystem genes can be upregulated after activation of the *lasRI* gene.[33,34] Our findings reveal that CDB may be a potential quorum sensing inhibitor, which warrants further investigation.

P. aeruginosa exhibits niche-specific motility, which is a critical determinant of its pathogenic potential.[35,36] The locomotive behavior of bacteria is intrinsically linked to virulence, demonstrating phenotypic plasticity dependent on physicochemical cues: (i) flagellum-driven swimming in aqueous matrices, (ii) surfactant-mediated swarming across viscous interfaces, and (iii) type IV pilus-dependent twitching on solid substrates.[37] Mechanistically, the rotary flagellar apparatus propels aqueous-phase navigation, whereas surface-associated motility requires the coordinated action of flagella and retractile type IV pili for biofilm expansion and tissue colonization.[38] Bacterial motility assays revealed that CDB could inhibit the motility ability of *P. aeruginosa* isolates through swimming, swarming, and twitching motilities in a concentration-dependent manner.

Meanwhile, we found that CDB could downregulate the mRNA relative expression of *lasR, pslA, pelA, algD*, and *algU* genes at a concentration of 128 μg/mL CDB. While higher concentrations (eg, 512 μg/mL) could theoretically be tested, our goal was to prioritize clinically translatable doses. Due to the formulation barriers and metabolic clearance, bioavailable concentrations in tissues are substantially lower, thus results from in vitro testing may not fully replicate the pharmacological effects observed in vivo. Building on these findings, our subsequent investigation will employ a murine cutaneous wound infection model to systematically evaluate the in vivo anti-virulence efficacy of CDB against *P. aeruginosa*.[39] Crucially, elucidating the anti-virulence mechanisms of CDB, particularly its capacity to disrupt *P. aeruginosa* biofilm ontogeny, suppress virulence determinants (eg, elastase and pyocyanin), and interfere with quorum-sensing circuitry, constitutes the pivotal first step in deconstructing its multimodal antimicrobial action. This mechanistic blueprint will inform the development of phytotherapeutic strategies targeting pathogenicity rather than bacterial lethality.

Conclusion

In conclusion, CDB, which is one of the most precious traditional Chinese medicines, exerts the positive anti-virulence efficacy on *P. aeruginosa*. CDB could significantly reduce the biofilm formation by downregulating the mRNA relative expression of biofilm-associated genes *lasR, pslA, pelA, algD*, and *algU*. In addition, CDB could efficiently inhibit the motility ability of *P. aeruginosa* isolates through swimming, swarming, and twitching motilities in a concentration-dependent manner. Our findings elucidate novel therapeutic paradigms for *P. aeruginosa* wound infection management through mechanism-guided clinical deployment of CDB. The collective evidence suggests that CDB is a promising phytotherapeutic candidate capable of suppressing *P. aeruginosa* virulence determinants, thereby addressing the critical limitations of conventional antimicrobial approaches for *P. aeruginosa* wound infection management in clinical settings.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article, and further inquiries can be directed to the corresponding authors.

Ethical Statement

This study complies with the Declaration of Helsinki. All study protocols were approved by the Ethics Committee of the School of Medicine, Huzhou University. There are no studies with humans or animals performed by any of the authors in this article. Informed consent was waived because this study with observational nature mainly focused on bacteria and did no interventions to patients.

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Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas, took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work. Xiangkuo Zheng and Xinhua Qiang are joint corresponding authors.

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Disclosure

The authors report no conflicts of interest in this work.

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