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Inhibitory effects of zinc oxide nanoparticles and morin on virulence-associated gene expression in Staphylococcus aureus

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Abstract
Objective: The emergence of multidrug-resistant Staphylococcus aureus has prompted interest in antivirulence strategies that attenuate pathogenicity without exerting bactericidal pressure. This study evaluated the inhibitory effects of zinc oxide nanoparticles (ZnO-NPs) and the flavonoid morin, individually and in combination, on transcriptional regulation of key S. aureus virulence genes.
Methods: ZnO-NPs were synthesised and characterised using X-ray diffraction (XRD) and scanning electron microscopy (SEM). The reference strain S. aureus ATCC 25923 and multidrug-resistant clinical isolates were employed. Minimum inhibitory concentrations (MICs) were determined by broth microdilution following CLSI guidelines. Sub-MIC concentrations of ZnO-NPs, morin, and their combination were used to evaluate transcriptional changes in eta, seb, agrA, sarA, and RNAIII by RT–qPCR, with 16S rRNA as a housekeeping control.
Results: ZnO-NPs demonstrated high crystallinity and nanoscale morphology (70–92 nm). At sub-MIC concentrations, ZnO-NPs significantly suppressed seb (0.12-fold) and eta (0.42-fold). Morin strongly inhibited seb (0.05-fold) but paradoxically induced eta (6.82-fold). Ciprofloxacin also upregulated eta (2.97-fold). The ZnO-NPs + morin combination neutralised morin-induced eta upregulation (3.01-fold) and produced complete suppression of agrA transcripts, with marked inhibition of sarA and RNAIII.
Conclusions: ZnO-NPs and morin act synergistically to repress S. aureus virulence gene expression, particularly through suppression of global regulators and toxin genes. Unlike conventional antibiotics, this combination reduces pathogenicity without applying bactericidal selection pressure. These findings highlight ZnO-NP–flavonoid combinations as promising antivirulence therapeutics, warranting further in vivo and translational studies.

Keywords: Staphylococcus aureus; Zinc Oxide Nanoparticles; Morin; Antivirulence Therapy; Quorum Sensing

Plain English Summary

Staphylococcus aureus is a bacterium that causes many infections, from skin boils to life-threatening sepsis. Because it is becoming resistant to many antibiotics, researchers are exploring new ways to fight it. Instead of directly killing the bacteria, which can drive resistance, antivirulence therapies aim to “disarm” the bacteria by stopping them from producing toxins.

In this study, we tested zinc oxide nanoparticles (ZnO-NPs), which are very tiny particles with antibacterial properties, and morin, a natural plant compound. We studied their ability to reduce the activity of genes that control toxin production in S. aureus.

We found that ZnO-NPs alone reduced the expression of important toxin genes. Morin also reduced one toxin gene but surprisingly increased another, which could worsen the disease. However, when used together, ZnO-NPs and morin worked in synergy: ZnO-NPs cancelled morin’s negative effect, and together they strongly suppressed genes that regulate multiple toxins.

This suggests that combining ZnO-NPs with morin may provide a safer, more sustainable approach to controlling S. aureus infections. Instead of killing the bacteria outright, the combination weakens them, making them less harmful and potentially easier to clear with standard treatments.

Introduction

Staphylococcus aureus is a highly adaptable opportunistic pathogen responsible for a wide range of infections, from superficial skin lesions to pneumonia, endocarditis, and septicaemia (1). Its ability to cause disease stems from a diverse array of virulence determinants, including toxins, adhesins, and regulatory systems that tightly control gene expression (2). Among these, the accessory gene regulator (agr) quorum-sensing system and the staphylococcal accessory regulator (sarA) play pivotal roles in coordinating toxin production, including staphylococcal enterotoxin B (seb) and exfoliative toxin A (eta) (3, 4).

The growing prevalence of methicillin-resistant S. aureus (MRSA) and other multidrug-resistant strains underscores the limitations of conventional antibiotics (5, 6). Traditional antimicrobial strategies impose strong selective pressure, accelerating resistance. By contrast, antivirulence therapies seek to neutralise pathogenicity by interfering with virulence regulation while preserving bacterial viability, thereby minimising resistance development (7).

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Nanomaterials such as zinc oxide nanoparticles (ZnO-NPs) have emerged as promising agents with both antimicrobial and antivirulence properties. Their physicochemical features, including crystalline wurtzite structures and nanoscale dimensions, enable the generation of reactive oxygen species (ROS) and strong interactions with bacterial membranes and regulatory pathways (8, 9). In parallel, plant-derived flavonoids such as morin have demonstrated antimicrobial, antioxidant, and quorum-sensing modulatory effects (10, 11). Yet, their combined impact on S. aureus virulence regulation remains underexplored.

This study investigated the individual and combined effects of ZnO-NPs and morin on the transcriptional regulation of S. aureus virulence genes, including seb, eta, agrA, sarA, and RNAIII. We hypothesised that the ZnO-NPs–morin combination would synergistically attenuate virulence gene expression, offering a novel therapeutic approach to combating resistant S. aureus infections.

Materials and Methods

Study Design

This experimental study investigated the inhibitory effects of zinc oxide nanoparticles (ZnO-NPs) and morin, individually and in combination, on the expression of selected virulence-associated genes in Staphylococcus aureus. Gene expression was quantified using RT–qPCR following treatment at sub-inhibitory concentrations of the test compounds.

Bacterial Strains and Culture Conditions

Reference strain: S. aureus ATCC 25923 (American Type Culture Collection, Manassas, VA, USA) was used as the primary strain for transcriptional profiling due to its well-characterised genome and stable virulence expression.

Clinical isolates

Multidrug-resistant clinical isolates of S. aureus were included for MIC determination to ensure translational relevance. Strains were maintained at –80 °C in nutrient broth supplemented with 20% glycerol. For experiments, cultures were revived on Tryptic Soy Agar (TSA) and incubated at 37 °C for 18–24 h. Colonies were inoculated into Tryptic Soy Broth (TSB) and grown with shaking (180 rpm) until mid-logarithmic phase. Bacterial suspensions were adjusted to 0.5 McFarland standard (~1.5 × 10^8 CFU/mL) before use.

Preparation of Test Compounds

Zinc oxide nanoparticles (ZnO-NPs): Commercial ZnO nanoparticle powder (<50 nm, >99.5% purity; Sigma-Aldrich, USA) was used. A 1024 µg/mL stock suspension was prepared in sterile deionised water, sonicated for 30 min to reduce aggregation, and freshly diluted as required.

Morin hydrate: Morin hydrate (≥90% purity; Sigma-Aldrich, USA) was dissolved in dimethyl sulfoxide (DMSO) at 2048 µg/mL. Final DMSO concentrations were ≤1% (v/v), a level shown not to affect bacterial growth or gene expression.

Nanoparticle Characterization

Structural and morphological features of ZnO-NPs were confirmed before biological assays:

X-ray diffraction (XRD): Performed to confirm crystal structure and phase purity.

Scanning electron microscopy (SEM): Used to assess particle size and morphology.

ZnO-NPs exhibited nanoscale dimensions (70–92 nm) and a hexagonal wurtzite crystalline structure, consistent with previous reports (8, 9).

Determination of Minimum Inhibitory Concentration (MIC)

MICs of ZnO-NPs and morin were determined by broth microdilution according to CLSI guidelines (12). Twofold serial dilutions were prepared in cation-adjusted Mueller–Hinton broth in 96-well plates:

ZnO-NPs: 512–1 µg/mL

Morin: 1024–2 µg/mL

Each well was inoculated with ~5 × 10^5 CFU/mL and incubated at 37 °C for 18–24 h. MIC was defined as the lowest concentration with no visible growth. Controls included: growth control (bacteria only), sterility control (medium only), and solvent control (1% DMSO).

Sub-inhibitory concentrations (¼ MIC: ZnO-NPs 16 µg/mL; morin 64 µg/mL) were selected for gene expression assays to ensure bacterial viability and allow assessment of transcriptional modulation.

RNA Extraction and cDNA Synthesis

Cultures were grown to mid-log phase and treated with sub-MIC ZnO-NPs, morin, ZnO-NPs + morin, ciprofloxacin (positive control), or left untreated. After 6 h incubation, cells were harvested, lysed with TRIzol reagent, and pre-treated with lysostaphin for efficient cell wall disruption.

Total RNA was purified and treated with DNase I.

RNA integrity was verified by absorbance ratios (A260/A280 1.9–2.1; A260/A230 >2.0).

First-strand cDNA synthesis was performed using 1 µg RNA, random hexamers, and M-MLV reverse transcriptase.

Quantitative Real-Time PCR (RT–qPCR)

RT–qPCR was performed using SYBR Green Master Mix on a StepOnePlus Real-Time PCR system (Applied Biosystems). Cycling conditions: 95 °C for 2 min, then 40 cycles of 95 °C for 10 s and 60 °C for 30 s, followed by melt-curve analysis.

Target genes: seb, eta, agrA, sarA, and RNAIII.

Internal control: 16S rRNA.

Primer design: Specific primers (82–278 bp amplicons) were validated for efficiency and specificity. (Corrected from original: sed and hla primers were excluded, as they were not analysed in this study).

Relative expression levels were calculated using the 2^−ΔΔCt method, with untreated samples as calibrators.

Statistical Analysis

All assays were performed in triplicate with three independent biological replicates. Gene expression data were expressed as mean fold-change relative to the control. Statistical significance between groups was assessed using one-way ANOVA with post hoc Tukey's test (GraphPad Prism v9.0). A p-value <0.05 was considered significant.

Results

Nanoparticle Characterization

The physicochemical properties of the zinc oxide nanoparticles (ZnO-NPs) were first confirmed before biological assays. X-ray diffraction (XRD) analysis demonstrated that the synthesised particles crystallised in a pure hexagonal wurtzite structure, as indicated by the sharp reflections

corresponding to the (100), (002), and (101) planes. The absence of secondary peaks confirmed both phase purity and a high degree of crystallinity. These features are consistent with ZnO nanoparticles reported to possess strong reactive surface activity (8, 9). The diffraction profile is shown in Figure 1.

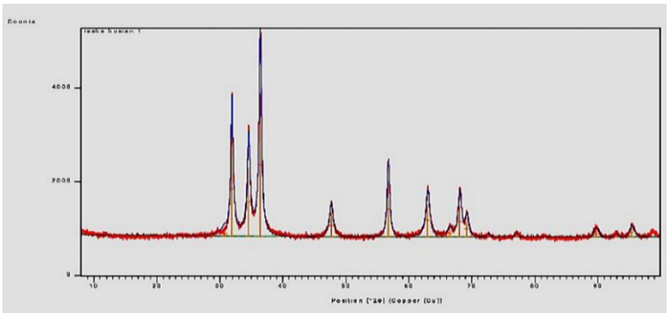

Figure 1: XRD profile of ZnO-NPs showing hexagonal wurtzite crystalline structure

Morphological assessment by scanning electron microscopy (SEM) revealed that the ZnO-NPs were uniformly distributed, with well-defined crystalline surfaces and particle sizes ranging from 70 to 92 nm. Such nanoscale dimensions provide a high surface-to-volume ratio that facilitates interactions with bacterial membranes and allows stable adsorption of small molecules such as morin. The SEM micrograph is presented in Figure 2. Collectively, these structural and morphological characteristics suggest that the ZnO-NPs were well suited for investigating biological interactions with S. aureus.

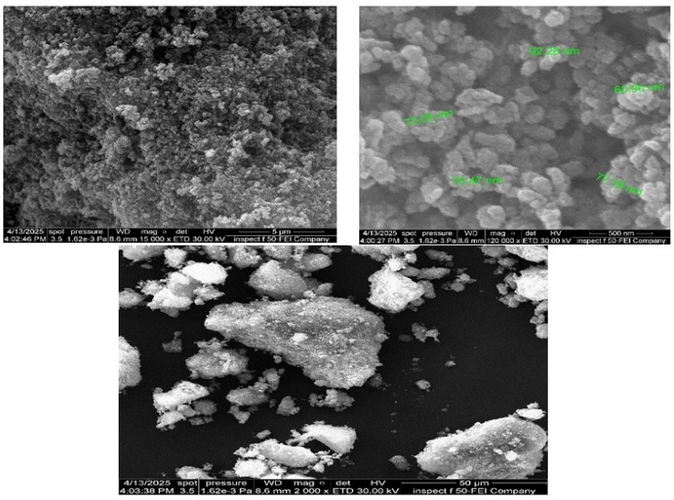

Figure 2: SEM image of ZnO-NPs demonstrating uniform nanoscale morphology (70–92 nm)

Minimum Inhibitory Concentrations

The antimicrobial activity of ZnO-NPs and morin was initially assessed using the broth microdilution method against S. aureus ATCC 25923 and multidrug-resistant clinical isolates. The minimum inhibitory concentration (MIC) of ZnO-NPs was found to be 64 µg/mL for the reference strain and 128 µg/mL for clinical isolates. For morin, MIC values were 256 µg/mL and 512 µg/mL, respectively. To ensure that gene expression was studied under non-lethal conditions, one-quarter of the MIC values (16 µg/mL for ZnO-NPs and 64 µg/mL for morin) were selected for subsequent experiments. These concentrations preserved bacterial viability and allowed the specific evaluation of antivirulence effects independent of bactericidal activity.

Effects on Virulence Gene Expression

The impact of ZnO-NPs, morin, and their combination on the transcription of key S. aureus virulence genes was investigated by RT–qPCR. The results are summarised in Table 1 and visually represented as a heatmap in Figure 3.

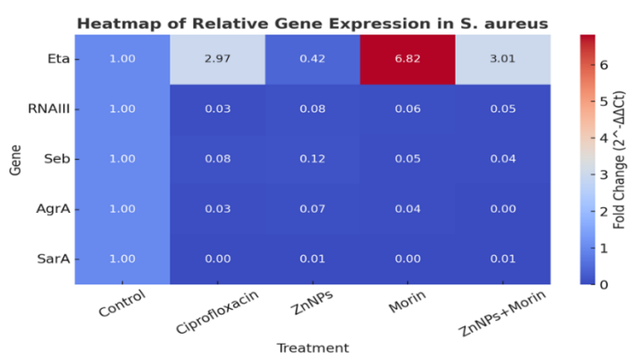

Figure 3: Heatmap illustrating relative expression levels of eta, seb, agrA, sarA, and RNAIII in S. aureus following treatment with ciprofloxacin, ZnO-NPs, morin, or their combination compared with untreated control

Treatment with ZnO-NPs alone led to significant repression of both toxin genes examined. Expression of seb was reduced to 0.12-fold relative to the untreated control ($p<0.01$), while eta was suppressed to 0.42-fold ($p<0.05$). These findings demonstrate that ZnO-NPs have a strong inhibitory effect on the transcription of major toxin genes.

In contrast, morin produced a mixed response. Although it markedly suppressed seb expression to 0.05-fold ($p<0.01$), it paradoxically induced eta expression to 6.82-fold ($p<0.001$). This unexpected stimulatory effect suggests that morin, while capable of repressing some virulence determinants, may also exacerbate others, raising questions about its suitability as a standalone antivirulence agent. Interestingly, ciprofloxacin, included as a conventional antibiotic comparator, also led to upregulation of eta (2.97-fold, $p<0.01$), underscoring the advantage of antivirulence therapies that avoid such paradoxical effects.

The combination of ZnO-NPs and morin produced the most pronounced overall inhibitory effect. While the eta induction caused by morin was not eliminated, it was significantly attenuated to 3.01-fold compared with morin alone ($p<0.05$). Importantly, the combination completely suppressed agrA transcripts (0.00-fold) and markedly inhibited sarA and RNAIII, reducing them to near-baseline levels. Because agrA and sarA are global regulators that control multiple downstream virulence genes, their suppression highlights the synergistic potential of ZnO-NPs and morin in disrupting quorum-sensing and toxin expression networks.

Table 1: Relative fold-change (2^−ΔΔCt) in expression of S. aureus virulence-associated genes following treatment (mean of three independent experiments)

Gene	Control	Ciprofloxacin	ZnO-NPs	Morin	ZnO-NPs + Morin
eta	1	2.97	0.42	6.82	3.01
RNAIII	1	0.03	0.08	0.06	0.05
seb	1	0.08	0.12	0.05	0.04
agrA	1	0.03	0.07	0.04	0
sarA	1	0	0.01	0	0.01

Summary of Findings

In summary, ZnO-NPs demonstrated broad inhibitory effects on both toxin and regulatory genes, whereas morin exerted selective inhibition accompanied by paradoxical induction of eta. Ciprofloxacin also upregulated eta, supporting the argument that traditional antibiotics may inadvertently enhance virulence. Most importantly, the combination of ZnO-NPs and morin not only suppressed most virulence determinants but also completely abolished agrA expression, indicating a synergistic antivirulence effect that could have translational relevance in developing alternative therapies against S. aureus.

Discussion

The present study investigated the antivirulence potential of zinc oxide nanoparticles (ZnO-NPs) and morin, individually and in combination, against Staphylococcus aureus. The findings demonstrate that ZnO-NPs significantly suppressed transcription of key virulence determinants, including seb and eta, and downregulated global regulators such as agrA, sarA, and their downstream effector RNAIII. Morin alone produced selective inhibition, notably reducing seb expression, but paradoxically upregulated eta, a toxin associated with exfoliative disease and increased pathogenicity. Importantly, the combination of ZnO-NPs and morin not only attenuated morin-induced eta activation but also achieved complete suppression of agrA, underscoring a synergistic antivirulence effect.

These results support the concept that combining nanoparticles with natural phytochemicals can achieve broader and more stable repression of bacterial virulence compared with either agent alone (13, 14, 15). Unlike bactericidal antibiotics such as ciprofloxacin, which also triggered upregulation of eta (16), ZnO-NP–morin combinations offer a non-lethal strategy that reduces pathogenicity without applying strong selective pressure for resistance development.

Comparison with Previous Studies

Several reports have highlighted the ability of ZnO-NPs to generate reactive oxygen species (ROS), disrupt bacterial membranes, and interfere with quorum-sensing pathways (8, 9, 17, 18). Our findings are consistent with these mechanisms, particularly the repression of seb and eta, which have previously been linked to ROS-mediated oxidative stress (19, 20). Similarly, morin has been shown to inhibit quorum sensing in other bacterial species (10, 11), although its paradoxical induction of eta observed here has not been widely reported. The mitigating effect of ZnO-NPs in the combination group suggests a mechanistic interplay, where nanoparticle-induced oxidative stress may buffer morin's undesired stimulatory influence.

The complete suppression of agrA observed in the combination treatment is particularly noteworthy. The agr system is a central quorum-sensing regulator in S. aureus and controls a broad network of virulence genes (3, 21). Its inhibition is associated with reduced toxin production, impaired biofilm formation, and enhanced immune clearance (4). By targeting this global regulator, ZnO-NPs and morin appear to synergistically dismantle the regulatory circuitry that underpins S. aureus pathogenicity.

Biological and Clinical Implications

The data highlight several important implications. First, antivirulence therapy provides a potential pathway for overcoming the limitations of conventional antibiotics. By disarming bacteria rather than killing them, such strategies may prolong the efficacy of existing antimicrobials and reduce the risk of resistance. Second, the paradoxical upregulation of eta by morin alone underscores the necessity of combinatorial approaches, as single agents may inadvertently worsen virulence profiles. Finally, the ability of the ZnO-NP–morin combination to simultaneously suppress toxins and quorum-sensing regulators suggests its promise as an adjunctive therapy to antibiotics, enhancing treatment outcomes without exacerbating resistance.

Study Limitations and Future Directions

This study has several limitations that must be acknowledged. First, gene expression analyses

were conducted exclusively on the reference strain ATCC 25923; validation in diverse clinical isolates is needed to ensure generalizability. Second, the experiments were limited to in vitro assays. Although sub-MIC exposures allowed evaluation of antivirulence effects without bactericidal pressure, in vivo studies are required to confirm safety, pharmacodynamics, and host immune interactions. Third, only a subset of virulence genes was examined. Comprehensive transcriptomic profiling would provide deeper insights into the breadth of regulatory disruption caused by the ZnO-NP–morin combination. Finally, statistical analyses were based on biological replicates, but the absence of detailed kinetic studies limits conclusions about the temporal dynamics of gene suppression.

Future research should therefore explore in vivo infection models to determine therapeutic efficacy and toxicity, investigate broader transcriptomic responses, and assess the stability of these effects in biofilm-associated infections. Mechanistic studies focusing on the molecular interplay between nanoparticle-induced ROS and flavonoid signalling pathways could also clarify the basis of synergy.

Conclusion

This study demonstrates that zinc oxide nanoparticles and morin act synergistically to suppress the expression of critical virulence genes in Staphylococcus aureus. ZnO-NPs alone repressed both toxins and regulatory genes, while morin selectively inhibited seb but paradoxically induced eta. Their combination overcame this paradox, neutralised eta induction, and completely suppressed agrA, a global regulator of pathogenicity. Unlike ciprofloxacin, which also triggered toxin upregulation, the ZnO-NP–morin combination attenuated virulence without exerting bactericidal pressure. These findings highlight nanoparticle–flavonoid combinations as promising antivirulence therapeutics, warranting further in vivo and translational studies to advance their potential as adjunctive treatments for resistant S. aureus infections.

List of Abbreviations

ATCC: American Type Culture Collection
CFU: Colony Forming Units
CLSI: Clinical and Laboratory Standards Institute
DMSO: Dimethyl Sulfoxide
MIC: Minimum Inhibitory Concentration
qPCR: Quantitative Polymerase Chain Reaction
RNAIII: Regulatory RNA III
ROS: Reactive Oxygen Species
SEM: Scanning Electron Microscopy
TSA: Tryptic Soy Agar
TSB: Tryptic Soy Broth
XRD: X-ray Diffraction
ZnO-NPs: Zinc Oxide Nanoparticles

Declarations

Ethics Approval and Consent to Participate

Not applicable. This study used bacterial strains and did not involve human or animal subjects.

Consent for Publication

Availability of Data and Materials

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

ARH: Conceptualisation and Writing – Original Draft
ARH and SMH: Methodology, Data analysis, Writing – Review & Editing
SMH: Supervision
Both authors read and approved the final manuscript.

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