



Original Research Article

Effect of *Monoon longifolium* extract on quorum-sensing-regulated production of virulence factors in *Serratia marcescens*

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Abstract

Background: The rise of multidrug-resistant bacteria has intensified the need for alternative therapeutic approaches that suppress pathogenicity without promoting resistance. Disrupting quorum sensing, an intercellular signaling mechanism that coordinates virulence, is one such approach. This study evaluated the antibacterial and anti-virulence potential of selected plant extracts against *Serratia marcescens*, with a focus on quorum sensing-regulated traits.

Materials and Methods: Aqueous extracts of *Moringa oleifera*, *Coleus amboinicus*, *Monoon longifolium*, and *Hibiscus rosa-sinensis* were screened for antibacterial activity and minimum inhibitory concentration (MIC) was determined. Sub-MIC concentrations were used to assess effects on biofilm formation, prodigiosin production, motility, and extracellular enzyme activity (lipase and protease). Phytochemical profiling was performed to identify major bioactive constituents.

Results: Among the tested extracts, *Monoon longifolium* exhibited antibacterial activity with an MIC of 12.75 mg/mL. At 8.5 mg/mL, it inhibited more than 70% biofilm formation and over 80% prodigiosin production, along with clear inhibition of lipase activity. *Moringa oleifera*, although not antibacterial, completely inhibited prodigiosin synthesis at 3.1875 mg/mL without inhibiting growth. Both extracts reduced biofilm formation, motility, pigment production, and enzyme secretion at sub-MIC levels. Phytochemical analysis confirmed the presence of flavonoids, sterols, terpenoids, and alkaloids.

Conclusion: The findings highlight the potential of plant-derived aqueous extracts as eco-friendly agents for mitigating bacterial pathogenicity, providing support for their possible application in controlling biofilm contamination within food-processing environments.

Keywords: Antibiotic resistance, Quorum sensing, Anti-virulence, *Serratia marcescens*, *Monoon longifolium*, *Moringa oleifera*.

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1. Introduction

The rapid rise of multidrug-resistant pathogens is diminishing the usefulness of existing antibiotics, creating an urgent need for approaches that weaken bacterial pathogenicity rather than inhibit growth. Targeting quorum sensing (QS), the regulatory system bacteria use to coordinate virulence gene expression, represents one such strategy.¹ Because QS-mediated behaviors such as toxin production, biofilm formation, motility, and secretion of extracellular enzymes occur only when bacterial populations reach high densities, blocking this communication network can attenuate virulence while leaving cell viability largely unaffected.^{2,3} Importantly, interfering with QS pathways imposes minimal selective pressure, helping preserve both the host microbiome and the long-term effectiveness of therapeutic agent.^{4,5}

QS systems in Gram-negative bacteria rely predominantly on N-acyl homoserine lactones (AHLs) as signaling molecules that bind cognate receptors to activate virulence-associated genes.² Anti-virulence or quorum-quenching (QQ) approaches may involve blocking QS receptors, degrading or mimicking signal molecules, or interfering with downstream transcriptional activation.⁶ Since QS inhibitors (QSIs) suppress virulence without hampering bacterial growth, they can be combined with antibiotics to increase treatment efficacy, reduce required dosages, and minimize adverse effects.⁷

Plant-derived metabolites are increasingly recognized as effective QS disruptors, capable of attenuating motility, virulence, and biofilm development.^{8,9} Compounds such as flavonoids, alkaloids, terpenoids, and phenolic constituents are known to interfere with key signaling pathways that

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coordinate QS-regulated behaviors. For example, curcumin from *Curcuma longa* inhibits QS gene expression and biofilms in *Pseudomonas aeruginosa*, while pyrogallol from *Embllica officinalis* and flavonoids like quercetin and naringenin interfere with QS-regulated bioluminescence.^[10,11,12] Ajoene from garlic and catechins from green tea reduce motility and virulence, while furocoumarins from grapefruit, limonoids from sour orange seeds, and cinnamaldehyde inhibit biofilm formation.^{13,14,15}

Serratia marcescens is a Gram-negative, opportunistic pathogen from the *Enterobacteriaceae* family, commonly associated with nosocomial infections, particularly in intensive care units. It colonizes the digestive, respiratory, and urinary tracts and spreads via medical equipment, lotions, and healthcare workers' hands.¹⁶ It causes pneumonia, sepsis, meningitis, and urinary tract infections, especially in immunocompromised patients, and exhibits resistance to multiple antibiotics, complicating treatment¹⁷⁻¹⁹ *S. marcescens* produces several virulence factors, notably the pigment prodigiosin, which contributes to biofilm formation and surface attachment. These are regulated by QS via AHLs, which also control proteases, lipases, and hemolysins. Owing to its QS-regulated virulence and biofilm-forming capabilities, *S. marcescens* is a suitable model for evaluating plant extracts with antimicrobial and anti-QS properties as potential alternatives for managing multidrug-resistant infections.

2. Materials and Methods

2.1. Bacterial strain

Serratia marcescens (gifted by Dr. Vishwas Sarangdhar, Caius Lab, St. Xavier's College) is a mutant strain producing far more prodigiosin pigment than the wild type strain. The culture was grown on Nutrient agar slants at 30°C.

2.2. Extract preparation

Monoon longifolium and *Moringa oleifera* leaves collected from Malad, Mumbai, were washed, air-dried, and oven-dried (60°C) overnight. The dried leaves were powdered, sieved, and soaked in distilled water (10% w/v) overnight in a shaker incubator. The mixture was centrifuged and filtered through Whatman paper. The filtrate was concentrated in a 40°C water bath, supplemented with DMSO to prevent contamination, and stored at -20°C.

2.3. MIC determination

Detection of antibacterial activity of aqueous leaf extracts of *Moringa oleifera*, *Coleus amboinicus*, *Monoon longifolium* and *Hibiscus rosa-sinensis* against *S. marcescens* was performed by the agar well diffusion method using Mueller-Hinton agar plates. Based on the results obtained, the Minimum Inhibitory Concentration (MIC) of *M. longifolium* extract against *S. marcescens* was assessed using the broth microdilution assay described by Wiegand et. al. (2008).²⁰

Various concentrations of the extract (12.75 mg/mL, 8.5 mg/mL, 6.375 mg/mL, 4.25 mg/mL, 3.1875 mg/mL, 2.125 mg/mL) were prepared in sterile Luria-Bertani (LB) broth to a final volume of 200 µL per well in a sterile 96-well microtiter plate. Sterility and untreated growth controls were also kept in triplicates. An overnight *S. marcescens* culture grown in LB was adjusted to 1×10^8 CFU/mL and subsequently diluted 1:100 in sterile broth; 50 µL of this inoculum was added to each test well. Plates were incubated overnight at 25°C. MIC was the lowest extract concentration of plant extract that completely inhibited growth. Growth was measured as increase in Optical density at 600 nm, and was recorded using a microplate reader.

2.4. Assay for biofilm inhibition

The antibiofilm activity of *M. longifolium* extract was evaluated using the crystal violet method by Shastri et al. (2025) in sterile 96-well microtiter plates.²¹ Each well received 180 µL LB supplemented with 0.2% (w/v) glucose, followed by 20 µL of the bacterial suspension and the plant extract at MIC and sub-MIC levels (12.75 mg/mL, 8.5 mg/mL, 6.375 mg/mL, 4.25 mg/mL, 3.1875 mg/mL, 2.125 mg/mL). The contents were gently mixed and incubated at RT for 72 hours. After incubation, the medium was carefully removed, and the wells were rinsed with phosphate-buffered saline (PBS, pH 7). The attached biofilm was then stained with 0.1% crystal violet, and any unbound stain was washed away with PBS. The retained dye was solubilized with acetic acid, and its absorbance was measured at 595 nm.

2.5. Assay for prodigiosin inhibition

Prodigiosin inhibition assay was performed using the method by Wang et al. (2024).²² *S. marcescens* culture in logarithmic phase ($OD_{600} = 0.6-0.8$) was resuspended in LB broth (supplemented with sucrose and peptone), both with and without the *Monoon longifolium* extract (12.75 mg/mL, 8.5 mg/mL, 6.375 mg/mL, 4.25 mg/mL, 3.1875 mg/mL, 2.125 mg/mL) and incubated at 25 °C in a shaker incubator for 48 hours. After incubation, 5 mL culture was withdrawn, mixed with 4 mL methanol, and vortexed thoroughly for 2 minutes to extract prodigiosin. Further, the mixture was centrifuged at 6000 rpm for 10 minutes, and 800 µL of the resulting supernatant was mixed with 200 µL of acidified methanol (4:1 of 0.05 N HCl and methanol) and the absorbance was measured at 530 nm.

2.6. Assay for inhibition of proteolytic activity

Inhibition of proteolytic activity was evaluated according to the procedure described by AlShaikh-Mubarak et al., (2023).²³ Overnight cultures treated with different concentrations of extract were centrifuged at 7000 rpm for 20 minutes at 4°C to obtain the cell-free supernatant (CFS), which served as the enzyme source. The reaction mixture consisted of 500 µL of Azocasein (0.08% w/v prepared in 0.2 M potassium phosphate buffer pH 7.0) and 500 µL of CFS. This was incubated at 37°C for 30 minutes to allow protease

activity. Reaction was stopped by adding 1 mL of 10% (w/v) trichloroacetic acid, followed by placing the tubes on crushed ice for 1 hour to ensure complete protein precipitation. After centrifugation to remove undigested azocasein, the resulting supernatant was mixed with an equal volume of 1 N NaOH. Absorbance was then measured at 440 nm. Blank had only distilled water.

2.7. Assay for inhibition of lipolytic activity

Bacterial cultures grown overnight in LB broth containing MIC and sub-MIC concentrations of the plant extract (12.75 mg/mL, 8.5 mg/mL, 6.375 mg/mL, 4.25 mg/mL, 3.1875 mg/mL, 2.125 mg/mL) were centrifuged at 14,000 rpm for 20 minutes and this cell free supernatant was dispensed into wells on Tributyrin Agar plates and incubated at RT for 24 hours. The lipase inhibition was evaluated by measuring the diameter of the clear zones around the wells under a light source.

2.8. Inhibition of swarming motility

Overnight culture of *S. marcescens* in logarithmic phase ($OD_{600} = 0.6-0.8$) was point-inoculated onto the surface of soft nutrient agar plates, incorporated with and without plant extract and incubated at RT for 24 hours. The swarming motility on both treated and control plates was compared.

2.9. Phytochemical screening of the plant extracts

Phytochemical profiling of the extracts was performed using standard qualitative tests based on the methods of Kancherla et al. (2019) and Harborne (1984), with minor modifications.^{24,25} Each extract was examined for major phytochemical groups, including flavonoids, phenols, alkaloids, tannins, terpenoids and sterols. The presence of these compounds was confirmed by observing specific colour reactions or the formation of characteristic precipitates.

3. Results and Discussion

The aqueous leaf extracts of *Moringa oleifera*, *Coleus amboinicus*, *Monoon longifolium* and *Hibiscus rosa-sinensis* were screened for antibacterial activity against *S. marcescens* by the agar well diffusion method. Only aqueous leaf extract of *M. longifolium* showed a zone of inhibition (zone diameter = 14 mm).

3.1. Determination of MIC

The Minimum Inhibitory Concentration (MIC) of *M. longifolium* aqueous leaf extract against *S. marcescens*, determined using the broth microdilution method, was found to be 12.75 mg/mL. Ankwai et al (2023) evaluated the effect of aqueous extract of *M. longifolium* against *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *E. coli*.²⁶ They found the MIC to be at 100, 200 and 200 mg/ml for *E. coli*, *P. aeruginosa* and *S. aureus*, respectively. The antibacterial activity of *M. longifolium* leaf extracts against *S. marcescens* has not yet been studied before.

3.2. Inhibition of biofilm formation

The antibiofilm potential of the plant extracts was assessed by the crystal violet staining method in 96-well microtiter plates. Biofilm formation was inhibited in a dose-dependent manner (**Figure 1**): 71% at 8.5 mg/mL ($\frac{2}{3}$ MIC), 63% at 6.375 mg/mL ($\frac{1}{2}$ MIC), and ~50% at 4.25 mg/mL ($\frac{1}{3}$ MIC) compared to the untreated control, which showed no inhibition in biofilm formation. In a study by Savu et al. (2022) using *S. aureus*, methanolic leaf extract of *M. longifolium* (also known by its synonym *Polyalthia longifolia*) showed highest biofilm inhibition (99.5%) at 0.039 mg/mL and antibiofilm activity was even seen at sub-MIC concentration, one-fourth of the MIC.²⁷ However, no studies investigating the anti-biofilm activity of *M. longifolium* aqueous leaf extract on *S. marcescens* could be found.

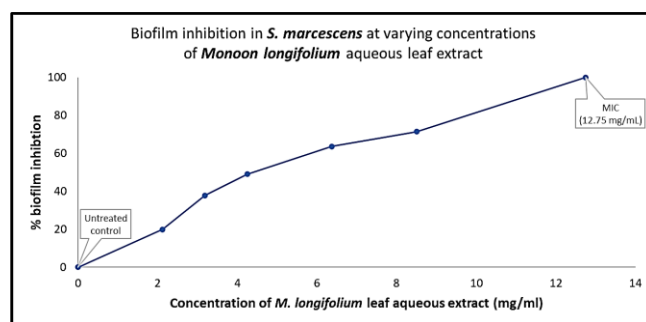


Figure 1: Biofilm inhibition in *S. marcescens* at varying concentrations of *Monoon longifolium* aqueous leaf extract.

In this study, the ability of plant extracts to prevent biofilm formation was examined on plastic surfaces (microtiter plates) and borosilicate glass surfaces (test tubes). However, in food-processing environments, biofilms can develop on a variety of surfaces, including plastic, glass, rubber, stainless steel and even food products.

3.3. Inhibition of prodigiosin production in *S. marcescens*

3.3.1. Qualitative assessment of prodigiosin inhibition

Prodigiosin inhibition was observed on gradient MHA plates supplemented with varying concentrations of the plant extracts. *S. marcescens* showed normal growth without pigment production at sub-MIC concentrations, indicating that sub-MIC levels are able to suppress virulence effectively without hindering bacterial viability (**Figure 2 A**). On the plates with MIC, no bacterial growth was observed in the region corresponding to the highest concentration. Aqueous extract of *Moringa*, which did not show any antibacterial activity against *S. marcescens* during the initial screening, shows evident inhibition of prodigiosin production at 3.1875 mg/mL (**Figure 2 B**).

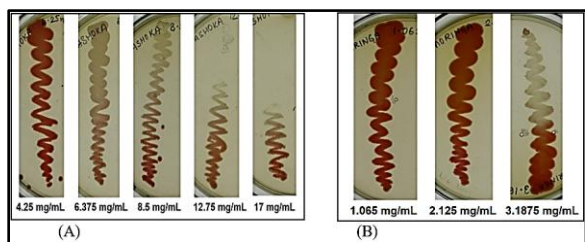


Figure 2: *S. marcescens* streaked on gradient MHA plates with various concentrations of aqueous leaf extracts of (A) *M. longifolium* (B) *M. oleifera*.

There are no studies available on the inhibitory effect of *M. longifolium* leaf extracts on prodigiosin production in *S. marcescens*. *M. oleifera* seed lectin was found to inhibit biofilm formation in *S. marcescens*,²⁸ but the inhibitory effects of *Moringa* aqueous leaf extract on prodigiosin production have not been studied.

3.3.1. Quantitative assessment of prodigiosin inhibition

Prodigiosin was extracted from *S. marcescens* after treatment with various concentrations of *M. longifolium* aqueous leaf extract for the quantitative assessment of prodigiosin inhibition. At 12.75 mg/mL, it showed 100% inhibition, while 80% inhibition was achieved at 8.5 mg/mL (Figure 3).

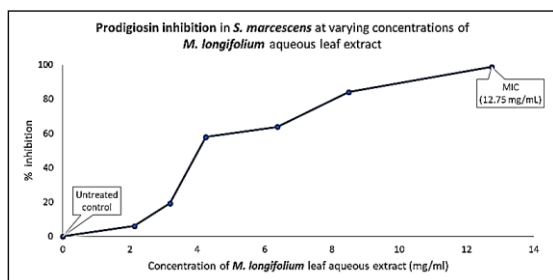


Figure 3: Prodigiosin inhibition in *S. marcescens* at varying concentrations of *Monoon longifolium* (Ashoka) aqueous leaf extract

3.4. Inhibition of lipase production in *S. marcescens*

Lipase activity, indicated by clear zones on tributyrin agar, was reduced in the presence of the plant extract, with noticeable inhibition at the MIC (12.75 mg/mL) and 2/3 MIC (8.5 mg/mL). However, no suppression of lipase production was observed at 1/2 MIC (6.375 mg/mL) or lower concentrations (Figure 4).



Figure 4: Lipid hydrolysis test of *S. marcescens* treated with various concentrations of *M. longifolium* aqueous leaf extract.

3.5. Inhibition of swarming motility in *S. marcescens*

The effect of *M. longifolium* aqueous leaf extract on swarming motility in *S. marcescens* was assessed by point-inoculating the bacteria on soft agar plates containing varying concentrations of the extract. In the untreated control (Figure 5 A), swarming occurred across the entire surface area of the agar plate. At concentrations of 4.25 mg/mL (Figure 5 B) and 6.375 mg/mL (Figure 5 C), swarming was progressively inhibited. At 8.5 mg/mL (Figure 5 D), there was a slight loss of pigmentation and complete inhibition of swarming. At concentrations of 10.625 mg/mL (Figure 5 E) both swarming and pigmentation were completely inhibited.

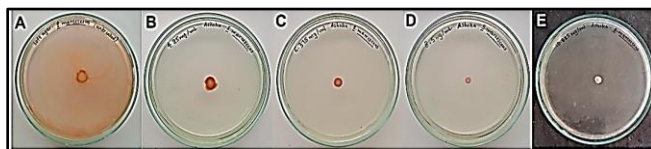


Figure 5: *S. marcescens* point-inoculated on soft agar plates (0.5% agar) with various concentrations of *M. longifolium* aqueous leaf extract. (A) Untreated control, (B) 4.25 mg/mL, (C) 6.375 mg/mL (D) 8.5 mg/mL, (E) 10.625 mg/ml.

Swarming motility plays an important role in surface colonization and biofilm formation. Eugenol, a phenolic compound found in cloves and cinnamon, inhibits swarming motility in *S. marcescens*. Swarming motility in *S. marcescens* is regulated by the flagellar master operon *flhD*.²⁹ The transcript level of *flhD* decreased more than 0.9-fold in *S. marcescens* ATCC 13880 upon eugenol treatment. No studies are available on the inhibitory effect of *M. longifolia* aqueous leaf extract on swarming motility in *S. marcescens*.

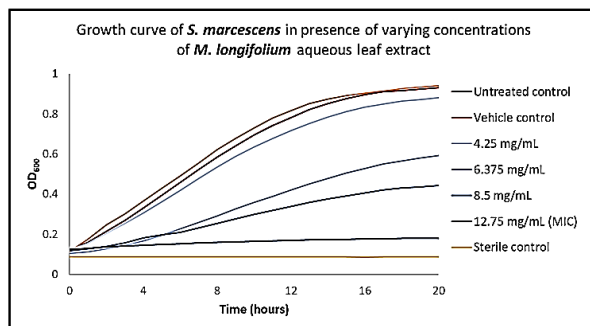


Figure 6: Growth curve of *S. marcescens* in the presence of varying concentrations of *M. longifolium* (Ashoka) aqueous leaf extract.

3.6. Growth curve analysis

Growth curve analysis showed that *M. longifolium* extract caused a gradual reduction in *S. marcescens* growth (Figure 6), eventually reaching complete inhibition at MIC (12.75 mg/mL). Considering the study’s focus on anti-virulence and QS inhibition as alternatives to conventional antimicrobials for addressing antimicrobial resistance (AMR), it is important to evaluate the potential of each extract to drive

resistance development. To contextualize these findings, **Figure 7** presents the growth curve of *S. marcescens* exposed to sub-MIC levels of ursolic acid, a pentacyclic triterpenoid known to inhibit QS in *S. aureus*, *C. albicans*, and *E. coli*.³⁰

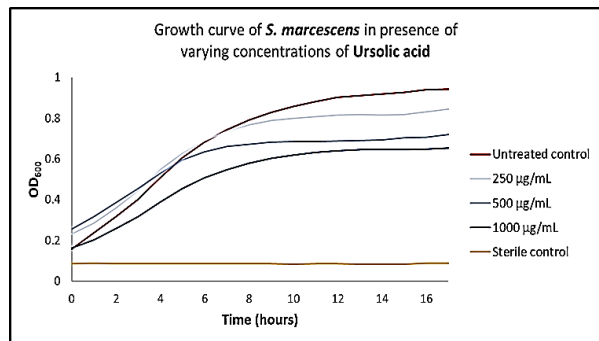


Figure 7: Growth curve of *S. marcescens* in the presence of varying concentrations of ursolic acid (known QS inhibitor).

3.7. Qualitative analysis of the phytochemicals in the plant extracts

Aqueous extract of *M. longifolium* (Ashoka) leaves contains flavonoids, sterols, terpenoids and alkaloids. These results confirm the findings by Soni et. al (2023) who showed the presence of glycosides, lyoniside, nudiposide, 5-methoxy-9-βxylopyranosyl, isolariciresinol, and schizandriside, and three flavonoids in the leaves of Ashoka.³¹ Aqueous extract of *M. oleifera* leaves contains flavonoids, alkaloids, terpenoids, sterols, tannins and phenols confirming the work done on aqueous extracts by El-Sherbini et al., (2024).³²

4. Conclusion

This study highlights the anti-QS potential of aqueous leaf extracts of *Monoon longifolium* and *Moringa oleifera* against *Serratia marcescens*, particularly through the inhibition of biofilm formation, prodigiosin production, lipase and protease activity, and swarming motility. While these results are promising, the extracts must be evaluated further using clinical and antibiotic-resistant isolates to determine their broader therapeutic relevance. Future work in quorum quenching can greatly benefit from isolating and characterizing the active constituents of plant extracts responsible for these effects. Techniques such as column chromatography can be employed to fractionate the extracts, enabling each fraction to be individually screened for anti-QS and anti-biofilm activity. Identifying the specific bioactive molecules will facilitate mechanistic studies and support the development of targeted anti-virulence agents. To better approximate biological conditions, these plant extracts should also be tested against established biofilms on mammalian epithelial tissues, such as mouse cell layers. This would help clarify whether the extracts can disrupt mature, structured biofilms, an essential feature for practical therapeutic use. Additionally, since biofilms commonly develop on diverse materials used in the food processing industry, including plastics, rubber, stainless steel, and glass,

future studies should assess efficacy of the extract on these surfaces. Collectively, these approaches could advance the discovery and development of plant-derived quorum-sensing inhibitors capable of mitigating bacterial virulence and persistent biofilms in both clinical and industrial environments.

5. Acknowledgement

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6. Conflict of Interest

The authors declare that they have no competing interests, financial or non-financial, related to this work.

7. Authors' Contribution

The first author is a student researcher who performed the experimental work and prepared the initial draft of the manuscript under the supervision of the corresponding author, who served as the research guide.

8. Source of Funding

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9. Conflict of Interest

None.

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