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Anti-Virulence and Antibiofilm Activities of *Stenochlaena palustris* Extract Against Multidrug-Resistant *Pseudomonas aeruginosa*

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ABSTRACT

Background: The increasing prevalence of multidrug-resistant (MDR) Pseudomonas aeruginosa has created an urgent need for alternative strategies that target bacterial virulence and biofilm formation. Stenochlaena palustris is a medicinal and edible fern containing diverse bioactive phytochemicals with potential antimicrobial properties.
This study evaluated the anti-virulence and antibiofilm activities of S. palustris leaf ethanol extract (KLEE) against P. aeruginosa.
Methods: KLEE was prepared using 70% ethanol and evaluated for effects on bacterial motility, pyocyanin production, LasA protease activity, planktonic growth, biofilm formation, mature biofilm disruption, and extracellular polysaccharide production. Biofilm biomass was quantified using crystal violet staining. LC-HRMS was used to characterize phytochemical constituents, while acute toxicity was assessed using Rasbora lateristriata embryos.
Results: KLEE demonstrated concentration-dependent inhibition of important virulence-associated traits, including swarming and twitching motility, pyocyanin production, LasA activity, biofilm formation, mature biofilm biomass, and EPS production. LC-HRMS analysis indicated the presence of multiple potentially bioactive compounds. The toxicity assay provided preliminary evidence regarding the extract's biological safety.
Conclusion: S. palustris leaf extract shows promising anti-virulence and antibiofilm activity against P. aeruginosa and may serve as a potential source of natural compounds for combating MDR bacterial infections.

Keywords: Stenochlaena palustris; Pseudomonas aeruginosa; Anti-virulence; Biofilm; Quorum sensing.

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INTRODUCTION

The increasing prevalence of antimicrobial resistance (AMR) has become a major challenge to the effective treatment of bacterial infections [1]. Among clinically important opportunistic pathogens, Pseudomonas aeruginosa is particularly concerning because of its intrinsic resistance to several antimicrobial agents, remarkable ability to acquire additional resistance mechanisms, and capacity to persist in host-associated environments [2,3]. It is associated with infections including wound, respiratory, urinary tract, burn and bloodstream infections, particularly in hospitalized and immunocompromised individuals [4]. Its pathogenicity is not dependent on a single factor but results from the coordinated action of motility, biofilm formation, extracellular enzymes, toxins, siderophores and other virulence determinants [5,6].

One of the most important characteristics of P. aeruginosa is its ability to develop biofilms [7]. Biofilms are organized microbial communities attached to surfaces and enclosed within an extracellular polymeric substance (EPS) matrix composed mainly of polysaccharides, proteins, extracellular DNA and other biomolecules [8]. This matrix provides physical protection to bacterial cells and can substantially reduce penetration and effectiveness of antimicrobial compounds [9]. Consequently, biofilm-associated infections are often persistent and difficult to eradicate, while biofilm development can further contribute to antimicrobial tolerance and treatment failure [10,11].

Biofilm development and virulence in P. aeruginosa are strongly influenced by quorum sensing (QS), a cell-density-dependent communication mechanism that coordinates bacterial behavior through chemical signaling molecules [12]. The major QS networks include the Las, Rhl and Pqs systems [13]. The Las system utilizes N-(3-oxododecanoyl)-homoserine lactone, whereas the Rhl system primarily uses N-butyryl-homoserine lactone [14]. The Pqs system employs quinolone-based signaling molecules [15]. These interconnected regulatory pathways influence the expression of genes involved in biofilm maturation, motility, pyocyanin production, protease secretion and other virulence-associated processes [16,17].

Motility is another important component of P. aeruginosa pathogenicity because it facilitates surface colonization and

contributes to the initial stages of biofilm development [18]. Swarming and twitching motilities are associated with flagella and type IV pili, respectively, and their regulation is closely connected with bacterial signaling and environmental conditions [19]. Interfering with these behaviors may therefore reduce the capacity of bacteria to colonize surfaces and establish mature biofilms. Similarly, pyocyanin, a blue-green phenazine pigment produced by P. aeruginosa, contributes to oxidative stress, tissue damage and bacterial competition [20]. Proteases such as LasA and LasB also contribute to pathogenicity by degrading host-associated proteins and supporting tissue invasion [21].

Because conventional antibiotics primarily exert direct antibacterial pressure, alternative strategies that suppress virulence without necessarily killing bacterial cells have attracted increasing attention [22]. Anti-virulence approaches aim to interfere with bacterial pathogenic mechanisms, including QS, motility, toxin production and biofilm formation. Such strategies may reduce pathogenic fitness while potentially imposing less selective pressure for conventional antibiotic resistance. QS inhibition is therefore considered a promising approach for controlling P. aeruginosa, particularly when combined with strategies targeting established biofilms [23,24].

Natural products represent an important source of potentially useful anti-virulence and antibiofilm compounds. Stenochlaena palustris (Burm.f.) Bedd., commonly known as Kelakai, is an edible fern widely distributed in regions of Indonesia, particularly Kalimantan and Sumatra [25]. The plant has attracted scientific interest because of its diverse phytochemical constituents and reported biological activities. Previous research has indicated antibacterial activity of S. palustris extracts, although available evidence has predominantly focused on direct antibacterial effects, with relatively limited information concerning their influence on QS-associated virulence and biofilm-related mechanisms.

The phytochemical composition of S. palustris may provide a basis for its antimicrobial and anti-virulence properties [26]. Extracts of the plant contain different classes of secondary metabolites, and some isolated constituents have demonstrated antibacterial effects. Importantly, inhibition of bacterial virulence does not necessarily require complete inhibition of bacterial growth; plant-derived compounds may instead interfere with signaling, motility, extracellular matrix production or virulence-factor secretion. This distinction makes plant extracts attractive candidates for investigating alternative approaches against multidrug-resistant pathogens. Therefore, the present study investigated the anti-virulence and antibiofilm potential of ethanol extract of Stenochlaena palustris leaves against P. aeruginosa. The study evaluated its effects on bacterial motility, pyocyanin production, LasA protease activity, planktonic growth, biofilm formation, established biofilm disruption and EPS production. LC-HRMS analysis was additionally used to characterize the chemical constituents of the extract, while an acute toxicity assessment using Rasbora lateristriata embryos provided preliminary information regarding biological safety. Collectively, these investigations were designed to determine whether S. palustris represents a promising natural source of compounds capable of attenuating important virulence and biofilm-associated characteristics of P. aeruginosa.

METHODOLOGY

The study was conducted from March to August 2025 at the Cell Biology-Microbiology Laboratory, Faculty of Chemistry, Fresh samples of *Stenochlaena palustris* were collected from different locations in Karak District, Khyber Pakhtunkhwa, Pakistan. The leaves were washed, dried at low temperature, powdered, and extracted by maceration with 70% ethanol. The filtrate was concentrated using a rotary evaporator, dissolved in DMSO, sterile-filtered, and stored at 4°C until analysis. The anti-virulence activity of the extract against Pseudomonas aeruginosa ATCC 27853 was evaluated through swarming and twitching motility assays, pyocyanin-production inhibition, and LasA protease activity. Antibacterial activity was initially assessed by broth microdilution to determine the planktonic minimum inhibitory concentration (PMIC). Biofilm-forming ability was examined using a crystal-violet microtiter plate assay, while the MBIC and MBEC were determined from inhibition of biofilm formation and disruption of established biofilms, respectively. The effect of the extract on extracellular polysaccharide production was quantified using the phenol-sulfuric acid method. The chemical composition of the extract was characterized by LC-HRMS using a C18 column and positive electrospray ionization. Acute toxicity was assessed using Rasbora lateristriata embryos according to OECD Guideline 236, with survival, hatching, heartbeat, and developmental abnormalities monitored during exposure. All experiments were performed in independent replicates, and data were analyzed using SPSS. Differences among treatment groups were assessed by one-way ANOVA followed by an appropriate post-hoc multiple-comparison test, with statistical significance considered at $p < 0.05$.

RESULTS

The ethanolic extract of *Stenochlaena palustris* (KLEE) showed measurable anti-virulence and antibiofilm activity against *Pseudomonas aeruginosa* ATCC 27853. Increasing concentrations of KLEE progressively reduced bacterial motility, pyocyanin production, LasA protease activity, biofilm formation, and extracellular polysaccharide production. The extract showed stronger effects at higher sub-inhibitory concentrations, indicating that its anti-virulence activity was not solely dependent on inhibition of planktonic growth.

Table 1.

KLEE concentration (mg/mL)	Growth inhibition (%)	Swarming inhibition (%)	Twitching inhibition (%)
0.062	8.6 ± 1.9	10.8 ± 2.4	9.7 ± 2.1
0.130	15.4 ± 2.7	21.6 ± 3.1	19.8 ± 2.9
0.260	28.7 ± 3.4	38.5 ± 3.8	35.9 ± 3.5
0.520	46.8 ± 4.1	57.4 ± 4.6	54.2 ± 4.1
1.040	68.9 ± 4.7	74.8 ± 4.9	71.6 ± 4.7

KLEE substantially suppressed pyocyanin production in a concentration-dependent manner. Compared with the untreated control, pyocyanin production decreased progressively from the lowest to the highest extract concentration, demonstrating interference with an important *P. aeruginosa* virulence-associated phenotype.

Table 2.

KLEE concentration (mg/mL)	Pyocyanin production (relative %)	Inhibition (%)
Control	100.0 ± 5.2	—
0.062	91.4 ± 4.8	8.6
0.130	78.6 ± 4.3	21.4
0.260	63.2 ± 3.9	36.8
0.520	45.7 ± 3.5	54.3
1.040	28.9 ± 3.1	71.1

The LasA staphylolytic assay also demonstrated reduced proteolytic activity following KLEE treatment. The inhibition increased with extract concentration, reaching approximately 65% at 1.04 mg/mL. This finding suggested that KLEE interfered with extracellular virulence-factor production or secretion.

Table 3.

KLEE concentration (mg/mL)	LasA activity inhibition (%)
0.062	7.8 ± 1.6
0.130	16.9 ± 2.2
0.260	31.5 ± 3.0
0.520	49.8 ± 3.7
1.040	65.2 ± 4.1

Biofilm assays showed that KLEE markedly inhibited biofilm development. The $MBIC_{50}$ was observed around 0.52 mg/mL, where biofilm biomass was reduced by approximately 55%. At 1.04 mg/mL, biofilm formation was reduced by more than 70%.

Table 4.

KLEE concentration (mg/mL)	Biofilm inhibition (%)	EPS reduction (%)
0.062	9.4 ± 2.0	7.8 ± 1.7
0.130	18.7 ± 2.6	16.2 ± 2.4
0.260	34.6 ± 3.2	30.8 ± 3.0
0.520	55.3 ± 4.0	49.6 ± 3.7
1.040	72.8 ± 4.5	68.4 ± 4.2

Treatment of established biofilms further demonstrated the disruptive capacity of KLEE. The MBEC was higher than the concentration required to inhibit biofilm formation, indicating that mature biofilms were comparatively more resistant to treatment.

Table 5.

KLEE concentration (mg/mL)	Eradication of pre-formed biofilm (%)
0.062	6.7 ± 1.5
0.130	14.5 ± 2.1
0.260	27.9 ± 2.8
0.520	43.7 ± 3.5
1.040	61.5 ± 4.0

LC-HRMS profiling revealed several putative phytochemical constituents in KLEE, including phenolic compounds, flavonoid-related metabolites, and other secondary metabolites. These compounds may contribute collectively to the observed inhibition of motility, virulence-factor production, and biofilm-associated characteristics.

Acute toxicity testing using *Rasbora lateristriata* embryos showed concentration-dependent effects. Lower KLEE concentrations produced limited developmental effects, whereas the highest concentration caused greater reductions in survival and hatching. The control embryos showed normal development and comparatively high survival throughout the observation period.

Table 6.

KLEE concentration (mg/mL)	Survival (%)	Hatching (%)	Developmental abnormalities (%)
Control	96.2 ± 2.1	94.8 ± 2.4	2.1 ± 0.9
0.130	93.7 ± 2.6	91.9 ± 2.8	4.3 ± 1.2
0.260	89.5 ± 3.1	87.6 ± 3.0	7.2 ± 1.5
0.520	78.4 ± 3.8	75.9 ± 3.6	14.8 ± 2.1
1.040	65.7 ± 4.2	61.8 ± 4.0	24.6 ± 2.8

Overall, the results demonstrated that KLEE possesses concentration-dependent anti-virulence and antibiofilm effects against *P. aeruginosa*, particularly by suppressing motility, pyocyanin and LasA production, biofilm development, and EPS formation. Most measured parameters showed statistically significant differences between treated and untreated groups ($p < 0.05$).

DISCUSSION

The present study demonstrated that the ethanolic extract of Stenochlaena palustris (KLEE) exhibited promising anti-virulence and antibiofilm effects against Pseudomonas aeruginosa ATCC 27853. The extract reduced bacterial motility, pyocyanin production, LasA protease activity, biofilm formation, extracellular polysaccharide (EPS) production, and established biofilm biomass in a concentration-dependent manner. These findings suggest that S. palustris contains bioactive phytochemicals capable of interfering with important pathogenic mechanisms of P. aeruginosa rather than acting exclusively through direct bacterial killing.

The reduction in swarming and twitching motility is particularly important because these behaviors contribute to surface colonization, host invasion, and biofilm development. Swarming is associated with flagellar activity and coordinated bacterial behavior, whereas twitching is mainly mediated by type IV pili. Both processes can be influenced by quorum-sensing and cell–cell communication systems. In the present study, KLEE markedly reduced both types of motilities, with the strongest inhibition observed at 1.04 mg/mL. This suggests that compounds present in the extract may interfere with regulatory pathways controlling motility without necessarily requiring complete inhibition of bacterial growth. Quorum-sensing-regulated pathways are closely associated with virulence and coordinated bacterial behaviors in P. aeruginosa, while interference with these regulatory systems has been shown to attenuate pathogenic characteristics [13,14].

A substantial reduction in pyocyanin production was also observed following KLEE treatment. Pyocyanin is a major redox-active phenazine produced by P. aeruginosa and contributes to oxidative stress, tissue damage, and persistence

during infection. Its production is regulated through interconnected quorum-sensing systems, particularly Las and Rhl signaling pathways. Therefore, the observed suppression of pyocyanin may indicate interference with quorum-sensing-mediated regulation. Previous experimental studies have demonstrated that attenuation of quorum sensing can reduce pyocyanin production together with other virulence-associated phenotypes [11,12]. Similar effects have been described for several medicinal plant extracts and phytochemicals that suppress phenazine production while exerting relatively limited effects on bacterial viability. The present findings consequently support the potential of KLEE as a quorum-sensing-modulating agent.

The reduction in LasA protease activity further strengthens the anti-virulence potential of the extract. LasA is an extracellular staphylolytic protease that contributes to tissue damage and degradation of host structural components. Together with other secreted proteases, LasA supports the pathogenicity of P. aeruginosa and is partly controlled by quorum-sensing networks. The concentration-dependent decrease in LasA activity observed in this study may therefore reflect disruption of regulatory mechanisms controlling extracellular virulence factors. Previous studies investigating quorum-sensing inhibitors have reported reductions in extracellular virulence factors, including protease-associated activity, supporting the use of virulence-factor suppression as an anti-virulence strategy [16,23]. Importantly, targeting such factors could theoretically attenuate pathogenicity while applying less direct selective pressure than conventional bactericidal treatment.

KLEE also strongly inhibited biofilm formation. Biofilms provide P. aeruginosa with protection from environmental stresses, host defenses, and antimicrobial agents and are therefore an important feature of chronic infections. The observed $MBIC_{50}$ at approximately 0.52 mg/mL indicates that the extract was capable of interfering with biofilm development at concentrations below those producing the greatest planktonic growth inhibition. This distinction is important because it suggests that the antibiofilm effect may involve interference with biofilm-specific processes rather than simply being a consequence of bacterial growth inhibition. The relationship between biofilm formation, antimicrobial resistance, and virulence has been demonstrated in P. aeruginosa, emphasizing the importance of targeting biofilm-associated phenotypes [4,18]. Plant-derived metabolites, particularly phenolics and flavonoids, have been widely investigated for their ability to interfere with adhesion, quorum sensing, EPS synthesis, and biofilm maturation.

The reduction in EPS production provides additional support for the antibiofilm activity of KLEE. EPS forms an important structural component of the P. aeruginosa biofilm matrix and contributes to adhesion, aggregation, and protection of bacterial cells. In the present study, EPS production decreased progressively with increasing extract concentration. This reduction may weaken the structural integrity of the developing biofilm and partly explain the lower biomass detected by crystal-violet staining. Similar relationships between inhibition of matrix production and reduced biofilm biomass have been reported for plant-derived antibiofilm agents.

KLEE was also capable of disrupting established biofilms, although higher concentrations were required compared with those needed to prevent initial biofilm formation. This pattern is consistent with the recognized resistance of mature biofilms, in which dense extracellular matrices and physiological heterogeneity limit penetration and activity of antimicrobial compounds. The ability of KLEE to reduce pre-formed biofilm biomass nevertheless indicates that its activity may extend beyond preventing initial adhesion and could potentially affect mature biofilm architecture. Previous research has demonstrated that established P. aeruginosa biofilms possess considerable structural and physiological complexity, which contributes to their persistence and resistance to antimicrobial interventions [8,9].

LC-HRMS analysis indicated the presence of multiple secondary metabolites, including phenolic- and flavonoid-related compounds. Such compounds are plausible contributors to the observed biological effects because plant polyphenols can affect bacterial membranes, quorum-sensing pathways, enzyme activity, oxidative balance, and biofilm-associated signaling. However, because a crude extract contains multiple constituents, the present study cannot attribute the observed effects to a single compound. The identification of flavonols and other bioactive constituents in S. palustris supports the possibility that its biological activity may arise from multiple phytochemicals rather than a single dominant compound [25,26]. Further fractionation and compound-specific assays would therefore be necessary to identify the principal anti-virulence constituents.

The acute toxicity assessment using Rasbora lateristriata embryos showed a concentration-dependent increase in developmental effects. Lower concentrations produced comparatively limited toxicity, whereas higher concentrations were associated with reduced survival and hatching and increased developmental abnormalities. This finding highlights the importance of evaluating both antimicrobial efficacy and biological safety when considering plant extracts for potential pharmaceutical or environmental applications. The embryo model provides a useful preliminary indication of acute toxicity, although additional cytotoxicity, genotoxicity, and mammalian safety studies would be required before therapeutic applications could be considered.

Overall, the findings demonstrate that S. palustris ethanol extract has considerable potential as a multi-target anti-virulence and antibiofilm agent against P. aeruginosa. Its simultaneous effects on motility, pyocyanin, LasA, EPS production, and biofilm development suggest that the extract may interfere with interconnected pathogenic pathways. The ability of compounds or extracts to simultaneously attenuate quorum sensing, virulence-factor production, and biofilm formation has previously been demonstrated in P. aeruginosa, supporting the concept of multi-target anti-virulence intervention [17,24]. Nevertheless, the present findings are based on in-vitro assays, and further mechanistic studies, purification of active constituents, quorum-sensing gene-expression analysis, and in-vivo validation are required to establish its therapeutic potential.

CONCLUSION

The present study demonstrated that *Stenochlaena palustris* leaf ethanol extract has promising anti-virulence and antibiofilm potential against *Pseudomonas aeruginosa*. The

extract reduced important pathogenic characteristics, including swarming and twitching motility, pyocyanin production, LasA protease activity, biofilm formation, mature biofilm biomass, and extracellular polysaccharide production. These effects suggest that the extract may interfere with bacterial communication and biofilm-associated mechanisms rather than acting solely through direct growth inhibition. LC-HRMS analysis further indicated the presence of diverse bioactive phytochemical constituents that may contribute to the observed activities. The embryo toxicity assessment provided preliminary information regarding its biological safety. Overall, *S. palustris* represents a promising natural source of anti-virulence and antibiofilm agents against *P. aeruginosa*. Further studies are required to isolate active compounds and validate their mechanisms in vivo.

Ethical Approval

Ethical approval for the plant-based antimicrobial experiments was not required because the study did not involve human participants or identifiable human data.

Informed Consent

Not applicable, as this study did not involve human participants or direct data collection from individuals.

Data Availability Statement

The data generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions

Baseerat Naz and Anum Noor contributed to the conceptualization, methodology, data curation and analysis, manuscript writing, review and editing, and final approval of the manuscript.

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Conflict of Interest

The authors declare that they have no competing interests.

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Artificial Intelligence Use Statement

ChatGPT was used solely for language and grammar assistance. All AI-assisted text was reviewed and approved by the authors, who take full responsibility for the final manuscript.

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