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Medicinal Plant-Derived Quorum Sensing Inhibitors: Mechanisms, Anti-Biofilm Activity, and Potential as Antibiotic Adjuvants

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Abstract: *Quorum sensing (QS) is a cell-density-dependent bacterial communication process that coordinates collective behaviours such as virulence-factor production , motility , secretion and biofilm development. Because these behaviours contribute to persistence and pathogenicity . QS has become an attractive target for anti-virulence research. Medicinal plants contain chemically diverse metabolites including flavonoids , phenolic compounds , terpenoids , tannins , coumarins and organosulfur constituents. Several of which have demonstrated anti-QS effects in experimental models. This review examines medicinal plant-derived quorum-sensing inhibitors (QSIs) with particular emphasis on Chromobacterium violaceum reporter assays and Pseudomonas aeruginosa phenotypes. The review discusses QS biology , mechanisms of inhibition , phytochemical classes , experimental assays , known medicinal plants , biofilm attenuation , antibiotic-adjuvant potential , limitations and translational challenges. Special attention is given to the methodological distinction between genuine QS interference and nonspecific antibacterial activity. Finally , Dolichandrone falcata is proposed as an underexplored candidate for QSI research because its phytochemistry and antimicrobial activity are documented , while a direct QSI study was not identified in the searches used for this manuscript. This research gap is presented as a hypothesis requiring experimental validation rather than as established activity.*

Keywords: QuorumSensing ; Quorum-Sensing Inhibitors ; Medicinal Plants ; Phytochemicals ; Pseudomonas aeruginosa ; Chromobacterium violaceum ; Biofilm ; Anti-virulence ; Antibiotic Adjuvant ; Dolichandrone falcata

I. INTRODUCTION

Antimicrobial resistance has created a need for therapeutic concepts that complement conventional antimicrobial action . One approach is anti-virulence therapy , in which pathogenic behaviour is weakened without necessarily relying on direct killing. QS is relevant to this strategy because bacterial populations use signalling molecules to coordinate collective responses. In pathogenic bacteria , QS can regulate secreted enzymes , pigments , motility , biofilm-associated functions and other traits that contribute to colonization and bacterial persistence.

P. aeruginosa is an especially important model because it combines biofilm formation , multiple virulence systems and clinically important antimicrobial resistance. Its Las , Rhl and PQS signalling networks interact to regulate numerous phenotypes. This makes QS inhibition an attractive research direction , but also means that a single phenotype cannot establish a complete mechanism.[1-3,12,13]

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II. SCOPE, RATIONALE AND LITERATURE APPROACH

The present review was developed as a narrative synthesis of pre-reviewed literature identified through PubMed , PubMed Central and publisher-indexed records. Search concepts included medicinal plants , quorum sensing , quorum-sensing inhibitors , quorum quenching , Chromobacterium violaceum , Pseudomonas aeruginosa , violacein , pyocyanin , biofilm , motility , phytochemicals and Dolichandrone falcata. Original studies were prioritized for plant-specific experimental claims , while review articles were used to establish mechanisms and broader context.The literature shows that plant-derived QS research has moved from simple reporter screening toward combined phenotypic and mechanistic approaches. Early screening studies evaluated multiple Indian medicinal plants using C. violaceum violacein production and P. aeruginosa motility , while later studies incorporated pyocyanin , biofilm , chemical profiling and computational analysis. For example , 24 Indian medicinal plants were screened in one study , while another evaluated 61 plants and identified Astilbe rivularis , Fragaria nubicola and Osbeckia nepalensis as particularly promising based on violacein inhibition without a detected growth effect.

The present review deliberately avoids treating every reduction in pigment or biofilm as proof of QSI. A plant extract may affect growth , membrane integrity , metabolism or stress responses , any of which can indirectly reduce a QS-associated phenotype. Therefore , the quality of evidence is considered alongside the reported activity.[1,3-8,12,13]

III. QUORUM SENSING: BIOLOGICAL BASIS

QS involves the production of signalling molecules , their accumulation in the environment , detection by bacterial regulatory systems and subsequent changes in gene expression. In many Gram-negative bacteria , AHLs function as signals. Other bacterial groups use different chemical signalling systems. The biological outcome is not simply a measurement of cell number ; signalling can synchronize behaviours across a population and thereby create coordinated phenotypes.

From a drug-discovery perspective , QS provides several possible intervention points. A compound may reduce signal synthesis , destroy or sequester a signal , prevent receptor recognition , alter downstream regulatory pathways , or indirectly suppress QS-regulated phenotypes. These mechanisms are not equivalent and require different levels of evidence.[1,2,12]

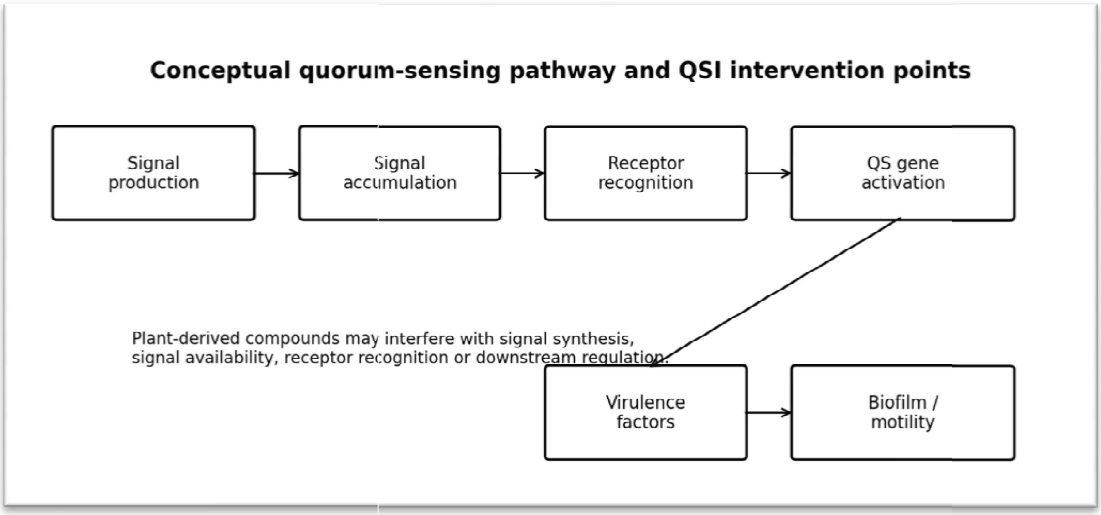


Fig. 1. Original schematic showing the conceptual QS pathway and possible intervention points for plant-derived QSIs.

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III. QUORUM-SENSING NETWORKS OF PSEUDOMONAS AERUGINOSA

P. aeruginosa possesses a complex QS network involving Las , Rhl and PQS-associated signalling. The Las system includes the 3-oxo-C12-HSL signal and LasR regulator ; the Rhl system includes C4-HSL and RhlR ; and the PQS system uses quinolone-associated signals. These systems are interconnected rather than functioning as isolated switches. Their collective effects influence virulence-associated factors , secondary metabolites , motility and biofilm behaviour. [2,11,13]

4.1 Las system

The Las system is often described as an upstream component of P. aeruginosa QS regulation. LasR activation can stimulate expression of multiple QS-related genes and influence production of extracellular virulence determinants. Interference with Las signalling has therefore been explored as one route to attenuate pathogenicity.[1,2,12,13]

4.2 Rhl system

The Rhl system is associated with C4-HSL signalling and regulates several phenotypes including rhamnolipid-associated functions and other QS-linked virulence traits. Because Rhl regulation interacts with other QS systems , effects on Rhl should be interpreted within the wider regulatory network.[1,2,11,13]

4.3 PQS system

The PQS system is linked to quinolone signalling and contributes to pyocyanin production , iron-related physiology and other aspects of P. aeruginosa behavior . Plant-derived compounds that alter PQS-associated phenotypes may therefore affect virulence through mechanisms beyond classical AHL receptor antagonism.[2,11,13]

4.4 Why P. aeruginosa is a useful QSI model

P. aeruginosa provides multiple measurable QS-associated endpoints. Pyocyanin, elastase , rhamnolipids , motility and biofilm formation can be studied alongside reporter systems. This multi-endpoint design is valuable because it reduces the risk of interpreting one nonspecific phenotype as definitive proof of QSI.[11-13]

V. CHROMOBACTERIUM VIOLACEUM AS A REPORTER ORGANISM

C. violaceum is widely used in preliminary QS screening because violacein production is controlled by QS in appropriate reporter strains. A reduction in violacein can be visually or quantitatively assessed. Studies of Indian medicinal plants have used C. violaceum strains to identify extracts with potential QS-interfering activity. [4,5,12,15].

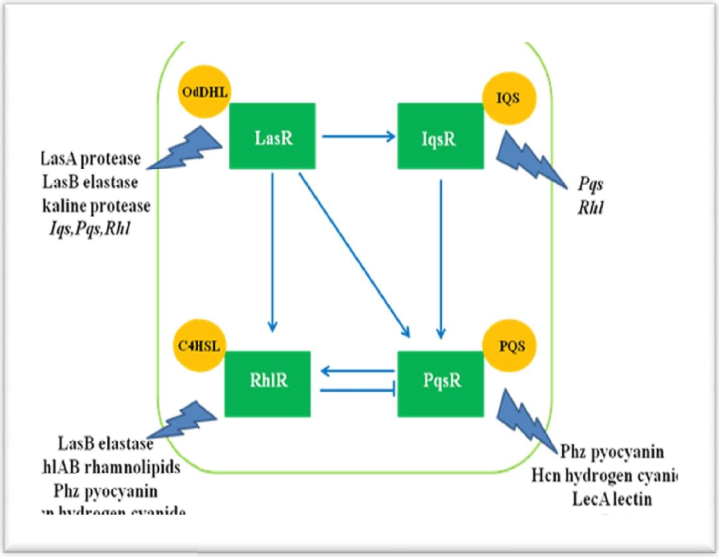


FIG. 2 : QUORUM SENSING NETWORK

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VI. MECHANISMS OF MEDICINAL PLANT-DERIVED QSI

Plant-derived phytochemicals

↓

1.Inhibit signal synthesis

→ Interfere with QS signal-producing enzymes

→ Confirm by signal quantification / enzyme studies

↓

2.Signal degradation / modification / sequestration

→ Reduce effective signal concentration

→ Confirm by chemical/biochemical evidence

↓

3.Receptor antagonism

→ Phytochemicals compete with or alter signal–receptor interaction

→ Docking : hypothesis , biological inhibition needs validation

↓

4. Downstream regulatory interference

→ Affect QS-related gene expression / c-di-GMP / stress pathways

→ ↓ Biofilm & virulence

↓

5. Multi-target activity

→ One phytochemical may affect signalling + membrane + metabolism + oxidative stress

→ Useful but mechanism is complex to assign.

[1-3,12,13]

VII. PHYTOCHEMICAL CLASSES

Reported anti-QS candidates include flavonoids , phenolic acids , phenylpropanoids , terpenoids , tannins , coumarins , quinones and organosulfur compounds. Examples discussed in the literature include eugenol , curcumin , ajoene and several flavonoids. Importantly , a plant containing a flavonoid is not automatically a QSI. Activity must be demonstrated in an appropriate model.[1,3,14,16-19]

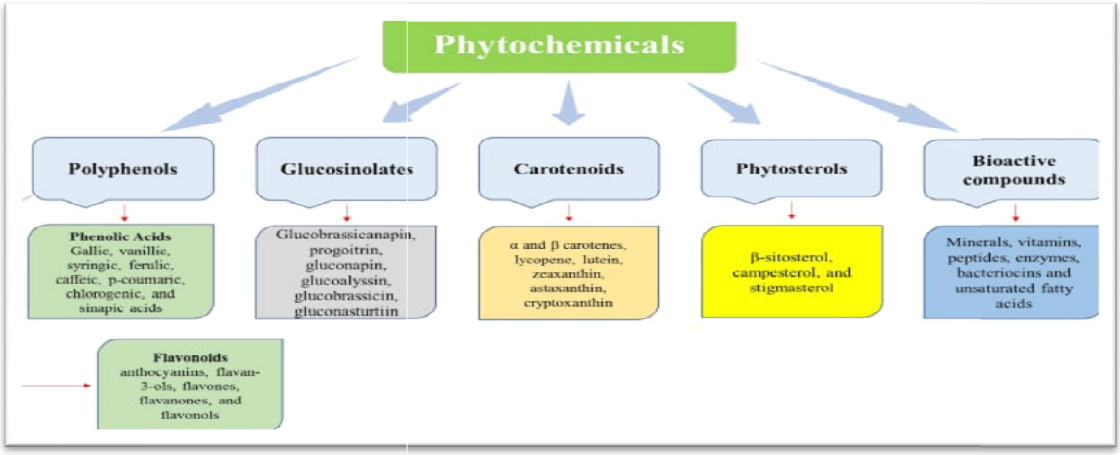


FIG. 3 : PHYTOCHEMICAL CLASSES OF QSI

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VIII. MAJOR PHYTOCHEMICAL GROUPS AND REPRESENTATIVE EVIDENCE

Class	Representative compounds	Potential QS-related effect
Flavonoids	Quercetin , naringenin , kaempferol , chrysin . [3,17–19]	Reporter/phenotypic inhibition ; receptor interaction proposed for selected compounds
Phenylpropanoids	Eugenol , cinnamaldehyde [17,18]	AHL/violacein-associated inhibition in selected models
Terpenoids	Carvacrol , linalool , phytol , limonene . [1,3]	Effects on biofilm , motility or virulence phenotypes
Organosulfur compounds	Ajoene and related garlic constituents [1]	Reduction of QS-regulated virulence and biofilm
Polyphenols/tannins	Hydrolysable tannins and related compounds [3,11]	Broad anti-QS activity in reporter systems
Diarylheptanoids	Curcumin [3]	Effects on pigment, virulence and biofilm phenotypes
Coumarins/quinones	Selected natural derivatives [19]	Potential signal/receptor and virulence modulation

Table 1. Major phytochemical groups and representative QS-related evidence [1,3,17–20].

IX. MEDICINAL PLANTS WITH EXPERIMENTAL QSI EVIDENCE

Plant	Experimental evidence	Interpretation
Hemidesmus indicus	Root extract inhibited violacein in C. violaceum screening [4]	Reporter-level evidence
Holarrhena antidysenterica	Bark extract inhibited violacein [4]	Reporter-level evidence
Mangifera indica	Seed extract inhibited violacein [4]	Reporter-level evidence
Punica granatum	Pericarp extract inhibited violacein [4]	Reporter-level evidence
Psoralea corylifolia	Seed extract inhibited violacein [4]	Reporter-level evidence
Astilbe rivularis	Leaf extract inhibited violacein; pyocyanin and swarming effects [5]	Multi-phenotype evidence
Fragaria nubicola	Leaf extract inhibited violacein; pyocyanin/swarming effects [5]	Multi-phenotype evidence
Osbeckia nepalensis	Leaf extract inhibited violacein; pyocyanin/swarming effects [5]	Multi-phenotype evidence
Citrus sinensis	Alcohol extract showed QSI; reduced pyocyanin/biofilm [6]	Multi-phenotype evidence
Laurus nobilis	Alcohol extract showed QSI [6]	Phenotypic evidence
Elettaria cardamomum	Alcohol extract showed QSI [6]	Phenotypic evidence
Allium cepa	Alcohol extract showed QSI [6]	Phenotypic evidence
Coriandrum sativum	Alcohol extract showed QSI [6]	Phenotypic evidence
Acacia nilotica	Pod fraction inhibited QS-associated virulence and biofilm [8]	Fraction + multi-phenotype evidence

Table 2. Medicinal plants with experimental quorum-sensing-inhibition evidence [4–8,11,12].

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X. EVIDENCE FROM INDIAN MEDICINAL PLANTS

Indian medicinal plants are particularly relevant to this research area because traditional pharmacopoeia provides a large candidate pool. A screening study of 24 medicinal plants identified Hemidesmus indicus , Holarrhena antidysenterica , Mangifera indica , Punica granatum and Psoralea corylifolia as extracts with varying violacein inhibition [4]. Another study screened 61 Indian medicinal plants and identified A. rivularis , F. nubicola and O. nepalensis as promising because their extracts reduced violacein without a negative effect on bacterial growth under the tested condition.

A separate study evaluated 20 medicinal plants and found strong quorum-quenching activity for extracts of Citrus sinensis , Laurus nobilis , Elettaria cardamomum , Allium cepa and Coriandrum sativum . These extracts also reduced pyocyanin production and biofilm formation in P. aeruginosa PA14 .

Tannin-rich medicinal plants provide another interesting group. A study of twelve Indian medicinal plants reported anti-QS activity in six extracts across subinhibitory concentrations , suggesting that tannin-rich preparations can contain molecules capable of interfering with signalling in both Gram-negative and Gram-positive models.[4,5,6,7]

XI. ANTI-BIOFILM ACTIVITY AND ITS RELATIONSHIP WITH QS

Biofilms are structured microbial communities embedded in extracellular material. They can display altered physiology and increased tolerance to environmental stress and antimicrobial exposure. QS contributes to biofilm-associated behaviour in many bacteria , including P. aeruginosa but QS is only one component of biofilm biology.

Plant-derived QSI research frequently reports reduced biofilm biomass after treatment. This is valuable because biofilm reduction can weaken persistence. Nevertheless , the interpretation must account for growth. If a concentration greatly reduces viable cells , a reduction in biofilm may simply reflect fewer cells. Strong studies therefore compare biofilm activity with planktonic growth and , when possible use microscopy or additional viability measures.[1,3,11,13,19]

XII. QS-ASSOCIATED VIRULENCE PHENOTYPE

Phenotype	Why it is useful	Main limitation
Violacein [4,5]	Convenient QS reporter endpoint	Sensitive to growth effects
Pyocyanin [6,11,13]	P. aeruginosa QS-associated pigment	Not exclusively controlled by one QS pathway
Elastase/protease [11,13]	Virulence-associated outputs	Can be influenced by general metabolic changes
Rhamnolipid [1,13]	Linked to QS and motility/biofilm behaviour	Complex regulation
Swarming [4,5,6]	Supports altered coordinated behaviour	Not QS-specific
Biofilm biomass [6,11,13]	Clinically relevant persistence phenotype	Many non-QS pathways affect it
Gene expression [13,17,19]	Can support pathway-level interpretation	Expression change does not always prove direct target binding

Table 3. QS-associated virulence phenotypes, their usefulness and interpretive limitations [1,2,11,13,17–19].

XIII. EXPERIMENTAL CONTROLS THAT STRENGTHEN A QSI STUDY

A credible QSI study should include untreated controls , solvent controls , bacterial-growth measurements and appropriate positive controls where available. Extract concentrations should ideally include a range below the MIC or below a defined growth-inhibitory threshold. Reporting only zones of inhibition is insufficient to establish QSI because agar diffusion primarily measures antibacterial effects.

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When a reporter strain is used , the study should distinguish pigment inhibition from loss of viability. Normalization to cell density , time-course analysis and independent phenotypes can greatly strengthen the interpretation.[4,5,7,11,13]

XIV. ANTIBIOTIC-ADJUVANT POTENTIAL OF QSIS

The concept of combining a QSI with an antibiotic is based on complementary actions. The antibiotic can target bacterial survival while the QSI may attenuate virulence or biofilm-associated behavior , Such combinations are attractive for research into difficult-to-treat infections , but the term synergy must be used carefully.

Quantitative combination studies should distinguish synergy from additivity , indifference and antagonism. A result observed at one concentration or one strain cannot automatically be generalized to all isolates. Plant extracts also require chemical standardization before combination findings can be reproduced.[3,11,13,14]

XV. RELATIONSHIP BETWEEN QS INHIBITION AND ANTIBIOTIC RESISTANCE

QS biofilm formation and antimicrobial resistance are interconnected but not interchangeable concepts. Biofilms can increase tolerance to antibiotics through physical barriers , altered growth states and microenvironmental effects. However , genetic resistance mechanisms such as target modification , drug inactivation and efflux may operate independently of QS.

Therefore , a QSI should preferably be described as an anti-virulence or biofilm-modulating candidate unless direct evidence demonstrates a defined effect on antimicrobial susceptibility or resistance-associated mechanisms.[11,13,14]

XVI. DOLICHANDRONE FALCATA: BOTANICAL AND PHARMACOLOGICAL BACKGROUND

Dolichandrone falcata . belongs to the family Bignoniaceae. A 2024 review reports its distribution in India, including Maharashtra , Goa, Karnataka and Bihar and describes a broad phytochemical and pharmacological literature. Reported constituents include dolichandroside A , chrysin, chrysin-7-rutinoside , rutin , pinocembrin , α-lapachone , lapachol , verbascoside and β-sitosterol together with broader classes such as flavonoids , terpenoids , alkaloids , polyphenols , saponins and steroids .Reported pharmacological properties include antimicrobial , anti-inflammatory , antidiabetic , antiulcer , antioxidant and other activities. Importantly for the present research question prior work has also examined D. falcata extracts against P. aeruginosa and other microorganisms , supporting its relevance as a candidate antimicrobial research plant.[9,10]



FIG. 4 : DOLICHANDRONE FALCATA

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XVII. WHY D. FALCATA IS A LOGICAL QSI CANDIDATE

Several features justify investigation without prematurely claiming activity. First, the plant contains chemically diverse secondary metabolites. Second , antimicrobial activity has already been reported. Third, several constituent classes found in the plant overlap with classes represented in the wider anti-QS literature. Fourth , D. falcata is available in Maharashtra , making authenticated plant collection more practical for a student research project.[1,3,9,10]

XVIII. RESEARCH GAP

The searches used for this review did not identify a direct experimental publication demonstrating D. falcata as a quorum-sensing inhibitor.[9,10]

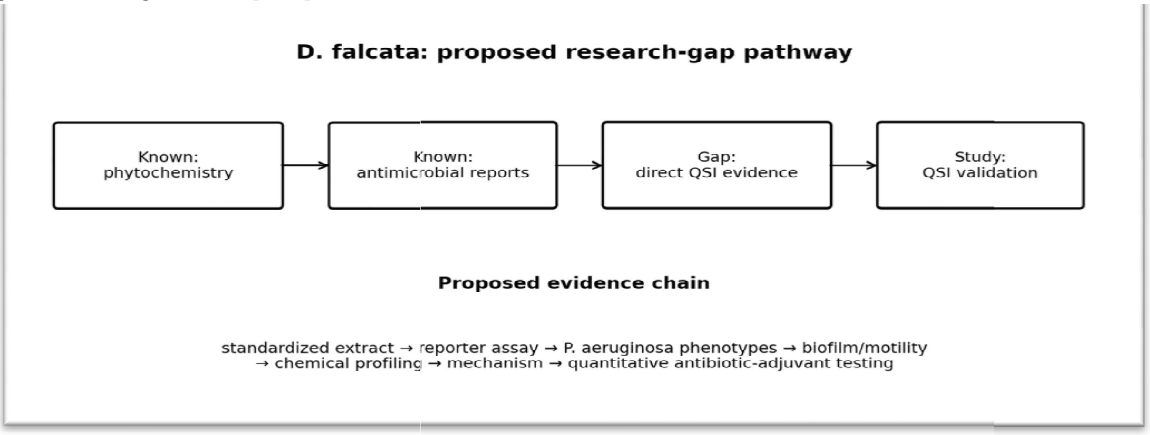


FIG.5 : RESEARCH HYPOTHESIS

XIX. PROPOSED RESEARCH FRAMEWORK FOR D. FALCATA

A scientifically defensible project can begin with authentication of the plant material and selection of a defined plant part , such as leaves. The extraction method should be standardized and recorded. Preliminary phytochemical screening and chromatographic profiling can provide a chemical baseline.The first biological stage can use a suitable C. violaceum reporter model to screen for reduction of violacein at concentrations that do not substantially inhibit bacterial growth. A positive result would justify progression to P. aeruginosa assays. The second stage can examine QS-associated phenotypes in P. aeruginosa, such as pyocyanin production , biofilm formation and motility. These endpoints should be measured alongside growth controls. If multiple phenotypes are altered without strong growth inhibition , the evidence for an anti-QS/anti-virulence effect becomes more persuasive.[4,5,8,9,11]

XX. POTENTIAL NOVELTY OF THE D. FALCATA PROJECT

The potential novelty of investigating Dolichandrone falcata lies not merely in selecting a medicinal plant source but in exploring its possible quorum-sensing inhibitory and anti-virulence properties through a systematic experimental approach. Although the plant has been investigated for its phytochemical constituents and various pharmacological activities, its potential role in quorum-sensing inhibition requires further systematic investigation. A comprehensive screening strategy involving multiple anti-virulence endpoints, antibiofilm evaluation and phytochemical profiling may provide valuable insights into its biological potential. Furthermore, investigating the relationship between phytochemical constituents and observed biological activities could help identify compounds associated with QSI-related effects. If supported by appropriate experimental evidence, evaluation of its potential as an antibiotic adjuvant could further strengthen its research significance. However, specific molecular targets or receptor-level interactions should not be claimed without appropriate mechanistic investigations.[8-10,13]

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XXI. ROLE OF PHYTOCHEMICAL PROFILING

Chemical profiling can help connect biological activity with candidate compounds. However , detection of a compound is not proof that it is responsible for the observed activity . Fractionation followed by activity-guided testing provides stronger evidence. If a purified compound reproduces the extract phenotype at relevant concentrations , the causal link becomes more convincing.[3,8,9]

XXII. FUTURE RESEARCH DIRECTIONS

- Standardized extraction and chemical fingerprinting across different geographical sources.
- Comparison of leaves, bark and other authenticated plant parts where scientifically justified.
- Fractionation guided by anti-QS activity rather than only total phenolic content.
- Use of more than one QS reporter or pathogen phenotype.
- Signal quantification to distinguish signal suppression from downstream effects.
- Genetic or biochemical validation of candidate QS targets.
- Evaluation of biofilm architecture and viability rather than biomass alone.
- Quantitative antibiotic-combination studies.
- Safety and cytotoxicity assessment of active fractions or compounds.
- Evaluation in more physiologically relevant models before any therapeutic claims.
- Investigation of formulation approaches for localized delivery if a strong candidate is identified.

[2,3,8,9,13]

XIII. CONCLUSION

Medicinal plants are promising sources of QSI and anti-virulence agents.
Studies report QS effects using C. violaceum and P. aeruginosa phenotypes such as pyocyanin , motility and biofilm.
Major gaps: growth effects , extract standardization and overinterpretation of docking results.
Strong research requires:
Authenticated plant → Standardized extraction → Sub-MIC testing → QS assays → Chemical profiling → Mechanistic validation.[1-3,5,11-13]

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