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Molecular detection of virulence genes in bacterial isolates from patients with bronchial asthma in Al-Anbar, Iraq

Virulence Genes in Asthma-Associated Bacteria

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Abstract

Objectives: This study aimed to isolate and identify bacteria from the respiratory tract of bronchial asthma patients and molecularly detect key virulence genes associated with these isolates.

Methods: A total of 168 samples were collected from patients with bronchial asthma. Bacterial isolates were identified using standard clinical microbiological methods. Polymerase chain reaction (PCR) was employed to detect the presence of virulence genes, including hlB (Staphylococcus aureus and S. epidermidis), ndvB (Escherichia coli), ureR (Proteus mirabilis), OmpA (Acinetobacter baumannii), Prot_clp (Klebsiella pneumoniae and K. oxytoca), lasB (Pseudomonas aeruginosa), and bibA (Streptococcus pneumoniae).

Results: Bacterial growth was obtained from all 168 samples. The distribution of virulence genes was species-specific: Prot_clp was detected in 100% (12/12) of K. pneumoniae and 40% (2/5) of K. oxytoca isolates. The lasB gene was present in 100% (17/17) of P. aeruginosa isolates. The ndvB gene was absent in all E. coli isolates (0/17). The OmpA gene was found in 76.5% (13/17) of A. baumannii isolates. The ureR gene was present in 100% (6/6) of P. mirabilis isolates. The hlB gene was confirmed in 100% (14/14) of S. aureus isolates but absent in all S. epidermidis isolates. Finally, the bibA gene was detected in 100% (10/10) of S. pneumoniae isolates.

Conclusions: Bacterial isolates from asthma patients harboured a high prevalence of specific virulence genes. These findings highlight the potential contribution of bacterial virulence factors to the pathophysiology of asthma and underscore the value of molecular methods in complementing traditional bacteriological diagnostics to better understand infection severity.

Keywords: Asthma, Virulence factors, PCR, Bacterial infection, Molecular diagnostics

Plain English Summary

This study looked at bacteria found in the lungs of people with asthma. The researchers wanted to know if these bacteria carried specific "virulence genes," which are like special weapons that help bacteria cause more severe infections. They collected samples from 168 asthma patients, grew the bacteria in the lab, and then used a genetic test (PCR) to check for these specific genes.

We found that the bacteria from asthma patients often carried these weapon-like genes. For example:

1. All of the Pseudomonas aeruginosa and Staphylococcus aureus bacteria had their specific virulence genes.
2. A high percentage of other common bacteria, like Acinetobacter baumannii and Klebsiella pneumoniae, also had them.
3. The presence of these genes usually matched what the researchers saw when they tested the bacteria's behaviour in the lab (e.g., whether they could break down blood cells or produce slimy biofilms).

This matters because asthma can be made worse by infections. If the bacteria living in the airways of asthma patients are equipped with these "weapons," they might be contributing to more severe or frequent asthma attacks. Knowing which virulence genes are present helps us understand how these bacteria might be making asthma worse.

Using genetic testing to find these virulence genes is a powerful tool that can be used alongside traditional lab methods. It gives doctors and scientists a clearer picture of the infection potential of bacteria in asthma patients, which could eventually lead to more targeted treatments.

Introduction

Asthma is a chronic inflammatory disorder of the airways characterised by variable airflow obstruction, bronchial hyperresponsiveness, and underlying inflammation (1). Clinical manifestations include recurrent episodes of wheezing, breathlessness, chest tightness, and coughing (2). The aetiology of asthma is multifactorial, involving complex interactions between genetic predisposition, environmental triggers, and immune responses.

Bacterial infections of the respiratory tract can significantly influence asthma pathogenesis and exacerbation. Certain bacteria can induce the release of histamine and other inflammatory mediators from mast cells and basophils, potentially triggering or worsening asthma symptoms (3, 4). Numerous bacterial species,

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including Klebsiella pneumoniae, Pseudomonas aeruginosa, Escherichia coli, Staphylococcus aureus, and Streptococcus pneumoniae, have been isolated from the sputum of asthma patients, suggesting a possible associative or causative role (5). The severity and persistence of these infections may be amplified by bacterial virulence factors—molecules that enable adhesion, invasion, immune evasion, and toxin production (6, 7).

While traditional culture methods identify causative organisms, they do not delineate pathogenic potential. Molecular detection of virulence genes offers a precise method to assess the arsenal of pathogenicity factors a bacterium possesses. This is crucial for understanding the mechanistic role of bacteria in asthma exacerbations and could inform more targeted therapeutic strategies.

This study aimed to:

1. Isolate and identify bacteria from patients with bronchial asthma in Al-Anbar Governorate, Iraq.
2. Molecularly detect a panel of virulence genes (hlB, ndvB, ureR, OmpA, Prot_clp, lasB, bibA) in the recovered isolates to assess their prevalence and potential clinical significance.

Materials and Methods

Study Population and Sample Collection

A cross-sectional study was conducted from April 15 to August 22, 2024. A total of 168 samples (sputum and endotracheal aspirates) were collected from patients diagnosed with bronchial asthma. Patients were recruited from Al-Ramadi General Teaching Hospital, Women and Children Hospital, and private respiratory clinics in Al-Anbar Governorate. Participants ranged in age from 5 to 70 years. Patients who had received antibiotic therapy within 72 hours before sample collection were excluded from the study. Diagnosis and confirmation of asthma were performed by specialist physicians. Clinical data for each patient were recorded using a standardised questionnaire. Ethical approval for this study was obtained from the Ethics Committee of the University of Anbar (Reference Number: #...). Written informed consent was acquired from all participants or their legal guardians.

Bacterial Isolation and Phenotypic Identification

Samples were cultured on Blood agar, Mannitol Salt agar, and MacConkey agar (HiMedia, India) and incubated aerobically at 37°C for 24-48 hours. Isolated colonies were sub-cultured to obtain pure isolates. Bacterial identification was performed based on Gram staining, colony morphology, and a series of biochemical tests, including catalase, oxidase, coagulase, IMViC (Indole, Methyl Red, Voges-Proskauer, Citrate), urease production, H_2S production on Kligler Iron Agar, and motility testing. Species-specific tests, such as growth on Cetrimide agar for P. aeruginosa, were also employed.

Phenotypic Detection of Virulence Factors

1. Hemolysin Production: Tested on 5% sheep blood agar. β-hemolysis was indicated by a clear zone around colonies, while α-hemolysis appeared as a greenish zone.
2. Protease Production: Assessed on skim milk agar. A clear halo around the bacterial growth indicated a positive result.
3. Urease Production: Detected using Christensen's urea agar. A colour change from yellow to pink indicated a positive result.
4. Pigment Production: P. aeruginosa isolates were tested for pyocyanin and pyoverdin production on Cetrimide agar.
5. Swarming Motility: Evaluated by inoculating P. mirabilis at the centre of a blood agar plate and observing for concentric rings of growth after incubation.
6. Biofilm Formation: Assessed using three methods: (a) Congo Red Agar (CRA) method (8), (b) Tube method (9), and (c) Microtiter plate (MTP) method (10).

Molecular Detection of Virulence Genes

1. DNA Extraction: Genomic DNA was extracted from pure bacterial colonies using the Genomic DNA Extraction Kit (Geneaid, Taiwan) according to the manufacturer's instructions.
2. Polymerase Chain Reaction (PCR): Specific primers for the target virulence genes (hlB, ndvB, ureR, OmpA, Prot_clp, lasB, bibA) were used (Table 1). PCR amplification was performed in a 25 µL reaction mixture containing 12.5 µL of EmeraldAmp Max PCR Master Mix (Takara, Japan), 1 µL of each forward and reverse primer (10 pmol/µL), 4.5 µL of nuclease-free water, and 6 µL of DNA template. The amplification conditions consisted of an initial denaturation at 95°C for 5 min; followed by 35 cycles of denaturation at 95°C for 45 s, annealing at a primer-specific temperature (52-60°C) for 45 s, and extension at 72°C for 1 min; with a final extension at 72°C for 7 min. The PCR products were separated by electrophoresis on a 1.5% agarose gel, stained with ethidium bromide, and visualised under a UV transilluminator.

Table 1: Primers used for the amplification of virulence genes

Gene	Target Bacteria	Product Size (bp)	Primer Sequence (5'–3')	Annealing Temp. (°C)	Reference
hlB	S. aureus, S. epidermidis	309	F: GTC CAC TTA CTG ACA ATA GTG C R: GTT GAT GAG TAG CTA CCT TCA GT	55	(10)
ndvB	E. coli	950	F: GGA CAG GGC AAG GTT TAT T R: GGT TAT ACT CAG CAG CAC TAT C	52	(11)
ureR	P. mirabilis	359	F: TGAGTGCGAAATTGCGATGG R: GCGGTTTATCACGAAGGGGT	58	(12)
OmpA	A. baumannii	578	F: GTTAAAGGCGACGTAGACG R: CCAGTGTTATCTGTGTGACC	60	(13)
Prot_clp	K. pneumoniae, K. oxytoca	602	F: TTGGAGGACCGCATCAT R: CTGCGGCTTGTTGATCTT	56	(14)
lasB	P. aeruginosa	300	F: GGA ATG AAC GAA GCG TTC TC R: GGT CCA GTA GTA GCG GTT GG	58	(15)

bibA	S. pneumoniae	389	F: AATCGAAAACAACGTTGGAAAG R: AAACCAGGCTTCATCAGTCATT	54	(16)

Statistical Analysis

Data were analysed using SPSS software version 26 (IBM, USA). Descriptive statistics were used to calculate frequencies and percentages of virulence factors and gene presence.

Results

Bacterial isolates were recovered from all 168 clinical samples collected from patients with asthma. The identified species included Klebsiella pneumoniae (n=12), K. oxytoca (n=5), Pseudomonas aeruginosa (n=17), Escherichia coli (n=17), Enterobacter cloacae (n=14), Acinetobacter baumannii (n=17), Proteus mirabilis (n=6), Staphylococcus aureus (n=14), Staphylococcus epidermidis (n=10), and Streptococcus pneumoniae (n=10).

The results of phenotypic virulence factor assays and molecular gene detection are summarised in Table 2.

Table 2: Summary of phenotypic and molecular virulence factor detection in bacterial isolates from asthma patients

Bacterial Species (n)	Haemolysis	Protease	Urease	Biofilm (MTP)	Virulence Gene (% Positive)
K. pneumoniae (12)	-	+	+	100%	Prot_clp: 100%
K. oxytoca (5)	-	+	+	100%	Prot_clp: 40%
P. aeruginosa (17)	β	+	-	100%	lasB: 100%
E. coli (17)	-	+	-	65%	ndvB: 0%
E. cloacae (14)	-	+	-	100%	-
A. baumannii (17)	-	+	-	100%	OmpA: 76.5%
P. mirabilis (6)	α	+	+	100%	ureR: 100%
S. aureus (14)	β	+	+	55.50%	hlB: 100%
S. epidermidis (10)	-	+	+	100%	hlB: 0%
S. pneumoniae (10)	α	+	-	100%	bibA: 100%

+ = positive; - = negative; α = alpha-haemolysis; β = beta-haemolysis

Hemolysin Production: β-hemolysis was observed in all isolates of S. aureus and P. aeruginosa. α-hemolysis was exhibited by P. mirabilis and S. pneumoniae. No hemolytic activity was detected for the other species.

Protease and Urease Production: All isolates tested positive for protease production. Urease activity was positive for K. pneumoniae, K. oxytoca, P. mirabilis, S. aureus, and S. epidermidis, and negative for the remaining species.

Pigmentation and Motility: All P. aeruginosa isolates produced pigments on Cetrimide agar (64% pyocyanin, 35% pyoverdin). All P. mirabilis isolates exhibited swarming motility.

Biofilm Formation: The Microtiter plate (MTP) method showed high biofilm-forming capacity (100% positivity) for most species, except E. coli (65%) and S. aureus (55.5%).

Molecular Detection of Virulence Genes: PCR results confirmed the high prevalence of specific virulence genes in their respective bacterial species, as detailed in Table 2 and Figures 1-8. Representative agarose gel electrophoresis images are shown below.

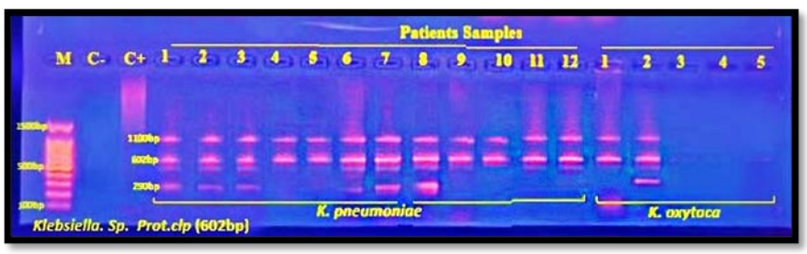

Figure 1: PCR amplification of the Prot_clp gene (602 bp) in K. pneumoniae (Lanes 1-12) and K. oxytoca (Lanes 13-17). Lane M: DNA molecular weight marker

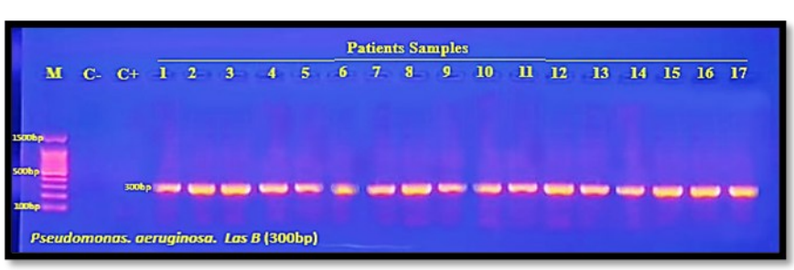

Figure 2: PCR amplification of the lasB gene (300 bp) in P. aeruginosa isolates (Lanes 1-17). Lane M: DNA molecular weight marker

Figure 3: PCR amplification of the ndvB gene (950 bp) in E. coli isolates (Lanes 1-17). No amplification observed. Lane M: DNA molecular weight marker; Lane PC: Positive control

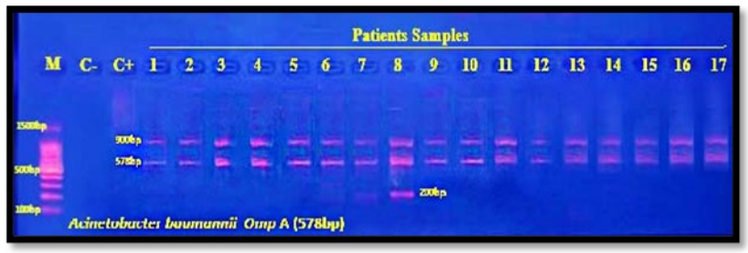

Figure 4: PCR amplification of the OmpA gene (578 bp) in A. baumannii isolates (Lanes 1-17). Positive isolates are shown. Lane M: DNA molecular weight marker

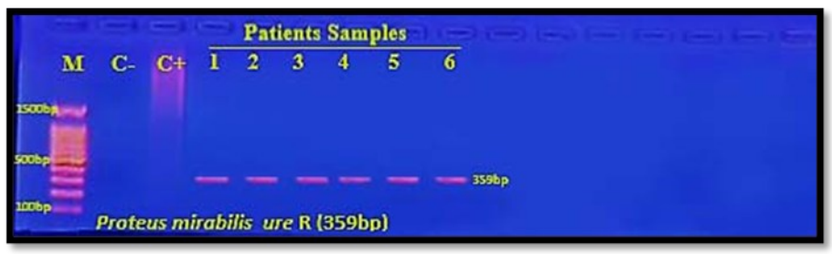

Figure 5: PCR amplification of the ureR gene (359 bp) in P. mirabilis isolates (Lanes 1-6). Lane M: DNA molecular weight marker

Figure 6: PCR amplification of the hlB gene (309 bp) in S. aureus isolates (Lanes 1-14). Lane M: DNA molecular weight marker

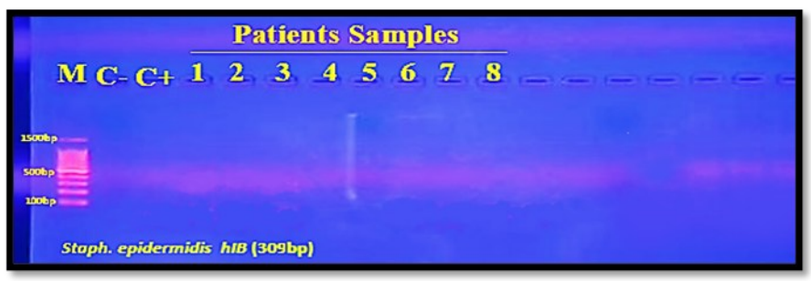

Figure 7: PCR amplification of the hlB gene (309 bp) in S. epidermidis isolates (Lanes 1-10). No amplification observed. Lane M: DNA molecular weight marker; Lane PC: Positive control

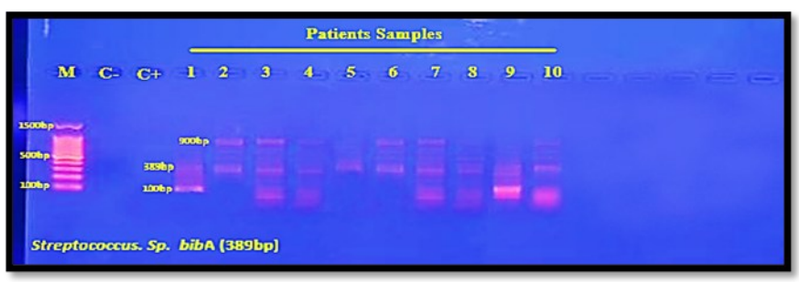

Figure 8: PCR amplification of the bibA gene (389 bp) in S. pneumoniae isolates (Lanes 1-10). Lane M: DNA molecular weight marker.

Discussion

This study provides a comprehensive analysis of virulence factors in bacteria isolated from the respiratory tract of patients with bronchial asthma. The 100% recovery rate of bacteria underscores their common presence in this patient population, consistent with previous studies (12, 13).

The phenotypic profiles aligned with expected bacterial characteristics. The species-specific hemolysis patterns (β-hemolysis in S. aureus and P. aeruginosa, α-hemolysis in P. mirabilis and S. pneumoniae) are well-documented virulence traits (14, 15). The universal production of protease among all isolates suggests a common mechanism for nutrient acquisition, tissue damage, and immune evasion (16). The detection of urease in Klebsiella spp., P. mirabilis, and staphylococci supports its role in host survival by modulating pH and generating ammonia (17).

The molecular findings are particularly significant. The high prevalence of the lasB gene (elastase) in all P. aeruginosa isolates (100%) is consistent with reports by Llanos et al. (18), who highlighted its critical role in lung tissue damage. The universal presence of the ureR regulator gene in P. mirabilis correlates with its strong urease phenotype, as noted by Ali and Maaroor (19). The absence of the biofilm-associated ndvB gene in all E. coli isolates was unexpected and contrasts with other studies (20); this may be due to strain-specific variations or the particular clonal lineages circulating in our patient population.

The high detection rate of the OmpA gene in A. baumannii (76.5%) confirms its importance as a key virulence factor involved in immune evasion, as supported by Azizi et al. (21). The species-specific presence of the hlB hemolysin gene in S. aureus and its absence in S. epidermidis is a key genetic marker differentiating these species, corroborating previous work by Fesharaki et al. (22) and Otto (23). The consistent detection of bibA in S. pneumoniae isolates aligns with its role in adhesion and pathogenesis (24).

Strengths and Limitations

The main strength of this study is the integration of phenotypic and genotypic virulence profiling across a diverse range of bacterial species from a substantial clinical cohort. However, several limitations must be acknowledged. The lack of a control group (non-asthmatic individuals) prevents conclusions about the specificity of these virulence factors to asthma. The cross-sectional design cannot establish causality or link specific virulence genes to asthma exacerbation severity. Furthermore, we detected the presence of genes but not their expression levels, which may more accurately reflect pathogenic activity.

Future Directions

Future research should involve longitudinal studies to correlate virulence gene profiles with clinical outcomes in asthma. Quantifying gene expression and integrating these data with antibiotic susceptibility testing would provide a more complete picture for guiding targeted therapeutic interventions.

Conclusion

This study demonstrates a high prevalence of specific virulence genes in bacterial isolates from patients with bronchial asthma. The strong concordance between phenotypic assays and PCR results validates the use of molecular methods as a powerful complement to conventional microbiology. Identifying these virulence determinants is a crucial step towards understanding the potential role of bacterial pathogens in asthma exacerbations. Ultimately, this approach could contribute to more nuanced diagnostic and therapeutic strategies aimed at managing bacterial infections in asthmatic patients.

List of Abbreviations

bibA: Bacterial immunogenic binding protein A gene (Streptococcus pneumoniae)
bp: Base pairs
CRA: Congo Red Agar
DNA: Deoxyribonucleic Acid
hlB: Haemolysin B gene (Staphylococcus aureus)
H_2S: Hydrogen sulfide
IMViC: Indole, Methyl Red, Voges-Proskauer, and Citrate utilisation tests
lasB: Elastase B gene (Pseudomonas aeruginosa)
ndvB: Nodulation protein B gene (Escherichia coli, biofilm-associated)
OmpA: Outer membrane protein A gene (Acinetobacter baumannii)
PCR: Polymerase Chain Reaction
Prot_clp: Protease Clp gene (Klebsiella pneumoniae, Klebsiella oxytoca)
ureR: Urease regulator gene (Proteus mirabilis)

Declarations

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the University of Anbar. Written informed consent was obtained from all participants or their guardians.

Consent for Publication

All authors have reviewed the final manuscript and provided consent for its publication under the Creative Commons Attribution Non-Commercial 4.0 International License.

Availability of Data and Materials

The datasets used are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare no competing interests.

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Authors' Contributions

MHH: Conceptualisation, Methodology, Investigation, Writing the Original Draft.
MFH: Supervision, Methodology, Writing - Review & Editing.
KSM: Supervision, Validation, Writing - Review & Editing.
All authors read and approved the final manuscript.

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