ORIGINAL ARTICLE

Molecular Genotyping of gyrA, blaOXA-51, and blaOXA-23 Genes in Multidrug-Resistant Acinetobacter baumannii Isolated from Childhood Asthma Patients in Iraq

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Key words:
Acinetobacter Baumannii, gyr a gene, oxa51 gene, oxa23 gene, and fluoroquinolone resistance

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ABSTRACT

Background*: Acinetobacter baumannii is a multidrug-resistant opportunistic pathogen implicated in hospital-acquired infections, particularly in immunocompromised populations. Its association with respiratory complications in childhood asthma poses significant treatment challenges owing to resistance mechanisms mediated by genes such as gyrA (fluoroquinolone resistance), intrinsic blaOXA-51 (a species marker), and acquired blaOXA-23 (carbapenem resistance).* ***Objective****:* ***The Aim of the present study*** *to isolate A. baumannii from pediatric asthma patients and characterize the prevalence of gyrA, blaOXA-51, and blaOXA-23 resistance genes.* ***Methodology****: A total of 250 sputum samples were collected from asthmatic children at the Al-Zahraa Teaching Hospital, Iraq (November 2024–April 2025). Isolates were identified using VITEK®2, CHROM agar, and biochemical tests. PCR amplification targeted gyrA, blaOXA-51, and blaOXA-23 genes. Antimicrobial susceptibility was assessed according to CLSI guidelines.* ***Results****: Thirty-six A. baumannii isolates were identified in this study. All the isolates (100%) harbored gyrA and blaOXA-51, whereas blaOXA-23 was detected in 22% of the isolates. High resistance rates (100%) were observed to cefotaxime, ertapenem, and colistin.* ***Conclusion****: The high prevalence of gyrA and carbapenems genes underscores the dominance of multidrug-resistant A. baumannii in pediatric asthma patients. These findings highlight the urgent need for molecular diagnostics and antimicrobial stewardship to mitigate treatment failures and nosocomial transmission.*

INTRODUCTION

Acinetobacter baumannii with asthma is one of the most significant opportunistic pathogens in healthcare settings, particularly among critically ill patients or those receiving prolonged medical treatments. *A. baumannii* known for its remarkable ability to survive on dry surfaces and to resist many classes of antibiotics, making it a major public health concern. Asthma, a prevalent chronic respiratory condition, manifests as cough, wheezing, dyspnea (shortness of breath), and chest tightness. As the underlying pathophysiology remains incompletely understood, further research is essential to discover improved therapeutics and biomarkers to enhance clinical outcomes [1,2]. One of the most concerning features of *A. baumannii* was increasing resistance to antibiotics, especially carbapenems, largely due to the presence of β-lactamase enzymes such as OXA-type carbapenems, including OXA-23 and OXA-51[3]. Previous studies have indicated that resistance severely limits treatment options and is associated with higher mortality rates in severe infections such as pneumonia and bloodstream infections [4].

Asthma is a chronic inflammatory disease of the airways and one of the most common respiratory diseases in children. Its symptoms range from mild coughing to severe shortness of breath, and are often caused by environmental or infectious factors. Recent studies have suggested that bacterial infections, particularly lower respiratory tract infections, may play a role in triggering or exacerbating asthma symptoms, particularly lower respiratory tract infections [5]. In a previous study, bacteria such as *A. baumannii* may contribute to the exacerbation of asthma, either through direct infection or exacerbation of chronic airway inflammation [6]. Therefore, the search for resistant bacteria in patients with asthma is essential to improve diagnosis and treatment outcomes. Although they are often linked to pathogenesis, certain microbes exhibit immunosuppressive capabilities with therapeutic potential. Both commensal and pathogenic bacteria show promise in modulating host interactions for clinical application. Reuter et al[7]. investigated microbial-based strategies as emerging treatments for inflammatory conditions by reviewing the current progress and future directions[7].

Several genes play key roles in both antimicrobial resistance and molecular diagnosis of *A. baumannii*. Among the bacterial species implicated in hospital-acquired infections, *A. baumannii* has emerged as a significant concern, particularly due to the rise of pan-drug-resistant (PDR) and Extensively Drug-Resistant (XDR) strains, which are notoriously challenging to eliminate[8]. *Acinetobacter baumannii* now exhibits resistance to a broad spectrum of antimicrobial agents, including fluoroquinolones (FQs), with PDR strains frequently contributing to the failure of antibiotic therapies [9]. The primary mechanism underlying FQ resistance involves spontaneous mutations within the quinolone resistance-determining region (QRDR) of the bacterial genome, specifically in the genes encoding DNA gyrase and topoisomerase IV. Mutations in the gyrA and parC subunits alter drug targets, thereby significantly reducing the efficacy of FQs and leading to high-level resistance[10]. Among these, gyrA, a housekeeping gene, has been commonly used as a molecular reference in genetic studies because of its relatively conserved sequence and its association with quinolone resistance when specific mutations occur[11]. Additionally, OXA-51 is a chromosomally encoded gene intrinsic to *A. baumannii* and is often used as a species-specific marker in molecular diagnostics [12]. In contrast, OXA-23 is typically plasmid-borne and plays a critical role in carbapenem resistance, with global dissemination posing a major threat to infection control [13]. The aim of the study to isolate and identify bacterial species from pediatric infections (sputum) with asthma and to determine the bacterial types associated with or contributing to asthma conditions. In addition, molecular detection of antibiotic resistance genes, such as (OXA-51 and OXA-23), which are known to play a major role in carbapenem resistance in *Acinetobacter baumannii*, to identify the housekeeping gene (gyrA), and use it as a molecular reference for bacterial identification and comparative genetic analysis among isolates.

METHODOLOGY

A total of 250 clinical sputum samples were collected from asthmatic patients during the period from November 2024 to April 2025 with hospital-acquired infections, such as AL-Zahraa Educational Hospital in Najaf, Iraq. 36 isolate of *Acinetobacter baumannii* were associated with hospital-acquired infections using Vitek diagnosis and polymerase chain reaction (PCR)-based amplification of DNA from a bacterial sample of childhood asthma infections.

CHROM AgarTM Acinetobacter Medium

Identification and Isolation: Initial isolation and presumptive identification were performed using CHROM agar medium (Chromagar, 1996), a selective formulation for gram-negative bacteria. plates were streaked with samples and incubated at 37 °C for 24 h. On this medium, both Acinetobacter spp. and Pseudomonas spp. formed red colonies. Differentiation between these genera was subsequently achieved using the oxidase test, in which Pseudomonas isolates were oxidase-positive and Acinetobacter isolates were oxidase-negative.

Confirmation with VITEK®2: Presumptive *Acinetobacter baumannii* isolates were confirmed using the automated VITEK® 2 compact system (BioMérieux) with GN ID cards. This card-based system identifies gram-negative bacteria by assessing their metabolic activity through 64 biochemical reactions, incorporating both established methods and novel substrates.

Phenotypic Characterization: All clinical specimens were initially cultured on nutrient agar (Oxoid Limited, England) to recover any bacterial pathogens. Recovered isolates were then characterized. MacConkey agar (Oxoid Limited, England) distinguished lactose-fermenting from non-lactose-fermenting organisms, while Herellea agar (Himedia, India) provided selective isolation for Acinetobacter spp. All plates were incubated at 37°C for 24h. The isolates were propagated and maintained following standard microbiological techniques[14]. For definitive phenotypic identification of *A. baumannii*, single colonies were subjected to several standard diagnostic tests. Typical gram-negative isolates were catalase-positive and citrate-positive and tested negative for both oxidase and indole production [15,16].

Antibiotic susceptibility

Following the Clinical Laboratory Standards Institute (CLSI, 2024) protocols [17], we employed the disc diffusion test to determine the susceptibility of isolates to several antibiotics: cefotaxime, novobiocin, amoxicillin, ertapenem, and colistin. The minimum inhibitory concentration (MIC) of ciprofloxacin was quantified using the E-test methodology (BioMérieux, France).

Molecular PCR technique

Chromosomal DNA extraction from gram-negative bacterial isolates was performed using the Wizard Genomic DNA Purification Kit, followed by the design of specific primers targeting the gyrA gene of *Acinetobacter baumannii* using bioinformatic tools and the NCBI database. Genotyping of *A. baumannii* isolates was conducted using three primer pairs: Gyr A (forward: GAAGTCAGGGCCGGTAATGT, reverse: TGTCGTTGGGGACGTAATCG; product size: 448 bp, designed in this study), OXA-51 (forward: TAATGCTTTGATCGGCCTTG, reverse: TGGATTGCACTTCATCTTGG; product size: 392 bp [18], and OXA-23 (forward: GATCGGATTGGAGAACCAGA, reverse: ATTTCTGACCGCATTTCCAT; product size: 465 bp [19]), which enabled the amplification and identification

of target genes associated with bacterial genotypic characterization.

Polymerase Chain Reaction Analysis

PCR amplification targeted the prevalent antibiotic resistance genes (*gyrA, Oxa51*, and *Oxa23*) to identify both wild-type sequences and mutations. Gene-specific primers (Table 1) were used along with the internal control primers. Each 50µl reaction contained: 1µg template DNA, 1µM of each primer, 200µM dNTPs, 1X reaction buffer, 1.5mM $MgCl_2$, and 1.5U Taq DNA polymerase. Thermocycling conditions were gene-dependent: *gyrA*: initial denaturation (95°C, 2 min); 30 cycles of [95°C (50 s), 57°C (50 s), 72°C (50 s)] *Oxa51*/*Oxa23*: Initial denaturation (95°C, 2 min); 30 cycles of [95°C (50 s), 54°C (50 s), 72°C (50 s)]; final extension (72°C, 5 min). Amplicons were electrophoresed on ethidium bromide-stained 2% agarose gels (20 µg/ml) and visualized under UV illumination[20].

Statistical analysis

Data processing was performed using bioinformatics tools and Microsoft Excel. Categorical variables were compared using a one-way similarity assessment and reported as percentages. Proportional differences were evaluated using the chi-square test, with statistical significance defined as *$p < 0.05$.

RESULTS

During the study period, 250 clinical sputum samples were collected from childhood asthma patients between November 2024 and April 2025 at Al-Zahraa Teaching Hospital in Najaf, Iraq. Thirty-six *A. baumannii* strains were isolated as opportunistic pathogens in pediatric asthma patients with hospital-acquired infections. Identification of colony morphology and microscopic and biochemical tests. On MacConkey agar, All *A. baumannii* colonies appeared small, pale yellow to pink, and non-partially lactose fermented. However, on CHROM agar, *A.baumannii* isolates appeared as red, round, smooth colonies (figure.1).

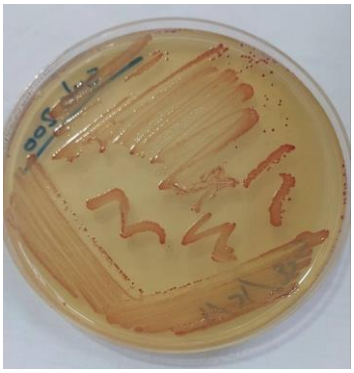

Fig. 1: Acinetobacter baumannii isolate on CHROM agar

Biochemical test results for *A. baumannii* isolates were obtained for urase, oxidase, simmone citrate, Kligler iron agar, and indole tests.

Table 1: Biochemical tests of *Acinetobacter baumannii* results

No	Biochemical tests	*Acinetobacter baumannii* results
1	Urase	Yellew / Yellew
2	Oxidase	+
3	Simmone citrate	Blue /blue
4	Kligler iron agar	Kla/ acid
5	Indole	+
6	Motilty	+

A. baumannii isolates (n=36) were cultured on MacConkey agar and identified using the VITEK® 2 GNID card system, which demonstrated a high identification accuracy (96-99% probability). Antibiotic susceptibility testing, performed according to the CLSI guidelines (Figure 2), revealed 100% resistance among these isolates to cefotaxime, novobiocin, amoxicillin, ertapenem, and colistin. This high prevalence of resistance to multiple drug classes indicates significant potential for the development of multidrug-resistant strains.

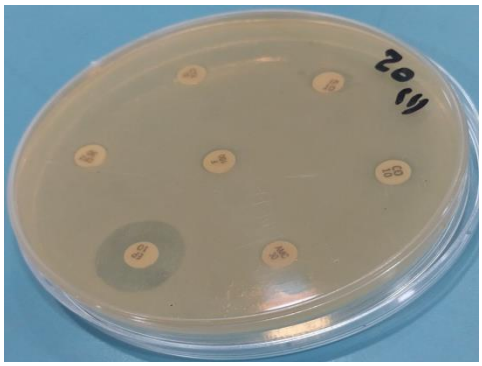

Fig. 2: Multidrug Resistance of *Acinetobacter baumannii* isolates for many antibiotics.

The present results indicate the presence of the gyrase subunit A (Gyr A) gene in Acinetobacter, which was detected in 36(100%) *A. baumannii* isolates. This unique gene encodes gyrase, an essential type II topoisomerase enzyme responsible for introducing negative supercoils into the DNA. Gyrases play a crucial role in DNA replication. A specific primer (*gyr A*) was designed using the online bioinformatics program of NCBI. PCR was used to detect the *(gyr A)* gene in *A. baumannii* [Table 1] and [Figures 3 and 4].

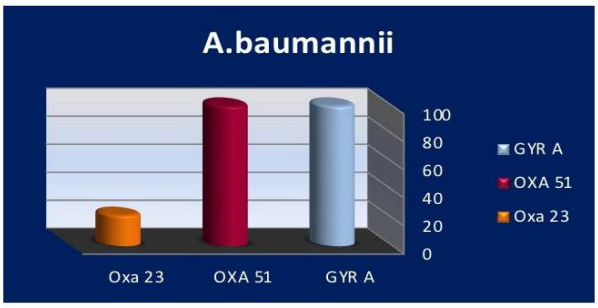

Fig. 3: prevalence of genes among *A. baumanii* isolated bacteria

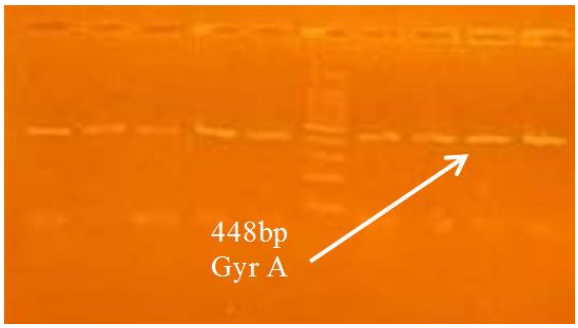

Fig. 4: The Agarose Gel Electrophoresis (2%) of PCR products of GyrA gene (448 bp) of *Acinetobacter baumannii* for (45) min at (80) volt.

The identification of the GyrA gene, these results support the prevalence of GyrA gene among *A. baumannii* isolates from children with asthma, highlighting a potential concern regarding antimicrobial resistance in this population. In contrast, the blaOXA-51-like gene was successfully amplified using PCR-specific primers for 36(100%) *A.baumannii* isolates that showed PCR amplification had specific produts (392bp) as shown in figures.3 and 5. The presence of these bands in all the tested isolates confirmed the detection of OXA-51, supporting the accurate identification of *A. baumannii* at the molecular level.

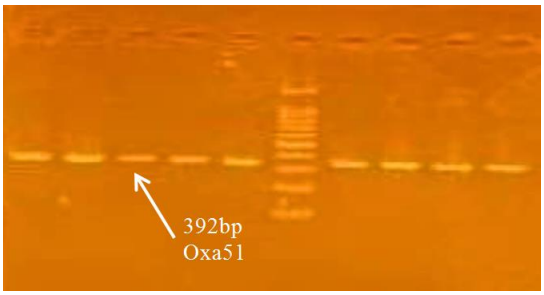

Fig. 5: The Agarose Gel Electrophoresis (2%) of PCR products of blaOXA-51 gene (392 bp) of Acinetobacter baumannii for (45) min at (80) volt.

The detection of the blaOXA-23 gene in *Acinetobacter baumannii* isolates from childhood asthma patients is a significant finding, and it revealed amplification of the blaOXA-23 gene in (22%) of *A.baumannii* isolates (figures3 and 6). Underscoring the prevalence of carbapenem-resistant strains in vulnerable populations. blaOXA-23 encodes a class D β-lactamase that confers resistance to carbapenem antibiotics and is often considered the last line of defense against multidrug-resistant infections.

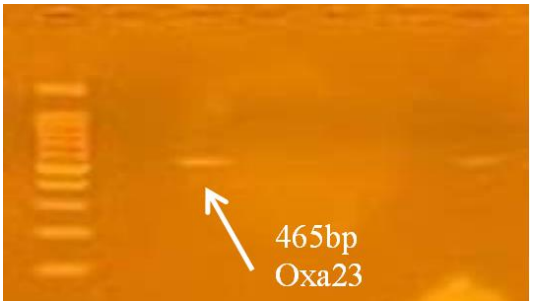

Fig. 6: The Agarose Gel Electrophoresis (2%) of PCR products of *blaOXA-23* gene (465 bp) of *Acinetobacter baumannii* for (45) min at (80) volt.

DISCUSSION

Identification of ***Acinetobacter baumannii*** according to morphology, biochemical tests, VITEK@2GN ID system, and molecular identification. On MacConkey agar, *A. baumannii* typically forms small, smooth, mucoid colonies that are pale or colorless because of their non-lactose-fermenting nature [21]. CHROM agar *A.baumannii* isolates appeared as round and smooth red colonies, which may develop a reddish-brown pigmentation over time, which could be attributed to prolonged incubation or specific strain variations[22]. These characters have led to its involvement in several nosocomial infections and outbreaks. It is a strict aerobic organism [23]. Similarly, a study published by Zhang et al [24]. investigated the relationship between glucocorticoid aerosol therapy and A. baumannii isolation in patients receiving invasive mechanical ventilation. The study found that treatment with aerosolized glucocorticoids was an factor for *A. baumannii* isolation, with a hazard ratio of 1.5, suggesting that independent risk corticosteroid use may increase the risk of *A. baumannii* colonization or infection in at-risk groups, such as children with asthma.

Antimicrobial agent's results reveled high resistance of *A. baumannii* isolates appeared high resistance 100% to cefotaxime, Novobiocin, amoxicillin, Ertapenem, and Colistin. These agents are known to significantly reduce the frequency of bacterial infections in pediatric asthma patients [25,26]. FQs represent the most frequently prescribed antimicrobial class in Iraq, and have historically demonstrated efficacy against *A. baumannii*

isolates within the country. However, resistance to these agents has emerged rapidly [27]. This trend is likely linked to the widespread availability of FQs across the multiple pharmaceutical markets in Iraq [28]. *Acinetobacter baumannii* has developed numerous acquired resistance mechanisms, enabling the expression of MDR, XDR, or PDR phenotypes. These highly resistant strains are associated with significantly increased morbidity and mortality [29–31].

Our results in the Molecular Characteristics Gyr A, bla-$_{Oxa}$ 51 and *bla*-$_{Oxa}$ 23 Genes in *Acinetobacter baumannii isolates* indicated that gyrA was present in 36(100%) of *A. baumannii* isolates. This unique gene encodes gyrase, an essential type II topoisomerase enzyme responsible for introducing negative supercoils into the DNA. The presence of gyrA in *A. baumannii* may pose significant therapeutic challenges in children with asthma, who are already at risk for respiratory impairment. In a previous study by Mohamed *et al.* [8], FQ resistance in *A. baumannii* isolates involved both target-site mutations and acquired PAFQR genes. Characterization of gyrA and parC sequences in the 12 selected isolates identified 12 distinct mutation patterns[8]. In Baghdad, Iraq, a study was conducted on 100 clinical samples of *A. baumannii* isolated from various sources, including blood, sputum, and burn wounds. PCR tests were used to detect gyrA, and 40% of the isolated samples were positive for this gene[32]. Detection of gyrA in several A. *baumannii* isolates from children with asthma highlights a potentially important resistance mechanism in this clinical population. GyrA encodes a subunit of DNA gyrase, a key enzyme that is targeted by fluoroquinolone antibiotics. Mutations in this gene have been well documented to contribute to fluoroquinolone resistance, which may significantly limit treatment options, especially in pediatric care where therapeutic options are already limited.

Our results for the blaOXA-51-like gene were successfully amplified using PCR-specific primers for 36(100%) *A.baumannii* isolates that showed PCR amplification with specific produts (392bp). The presence of bands in all the tested isolates confirmed the detection of OXA-51, supporting the accurate identification of *A. baumannii* at the molecular level. They reported that all 141 clinical isolates of *A. baumannii* tested positive for blaOXA-51-like, while 22 isolates of other Acinetobacter species did not carry this gene. This finding highlights the specificity of this gene and its usefulness in accurately identifying *A. baumannii* at the genetic level, thus enhancing the reliability of PCR-based diagnostic tools for clinical microbiology[24]. The presence of blaOXA-51-like alone in *Acinetobacter baumannii* does not necessarily confer high-level carbapenem resistance. However, its expression may be elevated in the presence of insertion elements, contributing to the emergence of resistance patterns. Therefore, while the presence of this gene confirms species identity, further investigation is needed to evaluate its role in resistance among clinical isolates, especially if patients show a poor response to standard treatments. This observation was supported by a study on ISAba1, which provides a promoter for blaOXA-like expression, investigating the role of ISAba1 in the expression of OXA carbapenemase genes in *A. baumannii*. Previous studies have found that among isolates containing a single carbapenemase gene similar to blaOXA-51, only those containing ISAba1, which is located adjacent to the gene, were resistant to carbapenems. This suggests that ISAba1 acts as an inducer of the blaOXA-51-like gene, enhancing its expression and contributing to its resistance[33].

The detection of the blaOXA-23 gene in *Acinetobacter baumannii* isolates from the sputum of childhood asthma patients is a significant finding, and it revealed amplification of the blaOXA-23 gene by (22%) of *A.baumannii* isolates (figures 3 and 6). The bla-OXA23 gene encoded another class D β-lactamase enzyme (OXA-23), which is strongly associated with acquired carbapenem resistance. A study published in Mosul, Iraq by Wajid et al. [34], identified blaOXA-23 in all *A. baumannii* isolates tested in clinical and hospital settings, indicating the high prevalence of this resistance marker in the region. Abdullah et al.[35], conducted a similar study in Baghdad. reported the presence of blaOXA-23 in a significant number of carbapenem-resistant *A. baumannii* isolates, highlighting its widespread prevalence. The presence of blaOXA-23 in pediatric patients is particularly concerning. Children with asthma are already at an increased risk of respiratory infections, and the emergence of multidrug-resistant pathogens further complicates the treatment options. In addition, Karampatakis et al [36]. focusing on pediatric care units, reported a predominance of *A. baumannii* strains producing both OXA-23 and OXA-58 carbapenemases, emphasizing the critical need for vigilant monitoring in such settings. **Conclusions:** This study highlights the clinical and microbiological significance of *Acinetobacter baumannii* in childhood asthma and demonstrates its potential role in respiratory infections and antibiotic resistance. Identification of the key resistance-associated genes blaOXA-51, blaOXA-23, and gyrA by PCR confirmed the presence of multidrug-resistant *A. baumannii* strains. The detection of blaOXA-23 in particular indicates widespread carbapenem resistance, whereas gyrA mutations suggest fluoroquinolone resistance, both of which complicate treatment strategies. The use of molecular diagnostic tools has provided reliable species confirmation and resistance profiling, emphasizing their utility in managing pediatric respiratory infections. These findings underscore the need for stringent infection control measures and careful antibiotic stewardship, especially in vulnerable pediatric populations with underlying respiratory conditions such as asthma.

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Ethics Approval and Consent to Participate: M.Sc. student, Adian Maytham Kazam Al-Khafaji Submitted the study was approved by the Faculty of Medicine, University of Kufa/Iraq. This study was supported by Dr. Hawraa Natiq Kabroot Al-Fatlawy. This article does not contain any studies involving human participants or animals performed by any of the authors.

Conflict of interest: The authors declare that there is no conflict of interest.

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Data Availability: All data analyzed in this study are included in the manuscript.

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