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DETECTION OF ADEB AND BASD GENES IN LUNG TISSUE SAMPLES OF RATTUS NORVEGICUS INJECTED WITH ACINETOBACTER BAUMANNII

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ABSTRACT
Acinetobacter baumannii is an opportunistic pathogen and a major cause of nosocomial infections, characterized by high resistance to multiple antibiotics. The success of its colonization and virulence is influenced by resistance factors such as the adeB gene and nutrient acquisition factors such as the basD gene. This study aimed to detect the presence of adeB and basD genes in lung tissue of Rattus norvegicus intravenously injected with clinical isolates of Acinetobacter baumannii. Six healthy female rats were divided into treatment groups with observation periods of 10 hours and 18 hours post-infection. Lung tissues were collected for DNA extraction, followed by PCR amplification using specific primers for adeB (549 bp) and basD (533 bp). Gel electrophoresis results showed that both genes were clearly detected in the bacterial isolate positive control, but not in lung tissue samples post-infection. The failure of detection in tissue samples is likely influenced by the low bacterial load, host-dependent regulation of gene expression, and the presence of PCR inhibitors derived from lung tissue. These findings indicate that although adeB and basD play crucial roles in resistance and virulence, their presence in host tissues is not always detectable by conventional PCR. Therefore, more sensitive detection methods are required for molecular analysis in in vivo models.

Keywords: acinetobacter baumanni; adeB gene; basD gene; lung tissue; PCR; rattus norvegicus

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INTRODUCTION

Acinetobacter baumannii is a Gram-negative bacterium recognized as a major opportunistic pathogen responsible for nosocomial infections. This bacterium exhibits remarkable survival abilities, even in environments that are hostile to most microorganisms, such as intensive care units (ICUs) and medical device surfaces (Ahmad et al., 2019; Almaghrabi et al., 2018; Zhao et al., 2023). Clinically, Acinetobacter baumannii can cause various nosocomial infections, including pneumonia, bacteremia, meningitis, soft tissue infections, and urinary tract infections (UTIs), particularly in immunocompromised patients. Transmission can occur rapidly via medical devices, potentially leading to systemic infections that are difficult to manage clinically (Ayoub Moubareck & Hammoudi Halat, 2020; Byun et al., 2021). Several studies have reported a high prevalence of Acinetobacter baumannii in respiratory tract infections; for instance, a study conducted at Haji Hospital Surabaya (2020–2022) reported that Acinetobacter baumannii ranked third among the most common causes of inpatient pneumonia, with a prevalence of 13.5% in 2020, increasing to 17.7% in 2022 (overall 15.7% of 185 isolates) (Akhmad et al., 2024).

One of the most severe forms of systemic infection caused by Acinetobacter baumannii is bloodstream infection (BSI). A study at RSUP Prof. Dr. I.G.N.G Ngoerah Denpasar in 2021 reported an Acinetobacter baumannii prevalence of 7.1% among 4,112 culture samples, with the majority of isolates obtained from endotracheal aspirates (30.5%) and blood (20.2%) (Yapson, 2023). Breslow et al. (2011) developed a systemic infection model of Acinetobacter baumannii in rats and observed bacterial colonization in lung tissue, accompanied by inflammation, edema, and

immune cell infiltration. These pathological changes resemble clinical manifestations in humans, such as pneumonia and sepsis. Such infections may progress to acute lung injury and systemic damage due to imbalances in reactive oxygen species (ROS). Rapid bacterial colonization triggers an excessive inflammatory response, leading to lung tissue damage, impaired oxygenation, respiratory failure, and premature death (Li et al., 2024).

A key mechanism underlying Acinetobacter baumannii antibiotic resistance is the AdeABC efflux pump system, composed of three components: AdeA, AdeB, and AdeC. Among these, the adeB gene plays a central role as a multidrug transporter capable of exporting various antibiotics out of the bacterial cell, thereby reducing therapeutic efficacy (Nazarov et al., 2022; Xu et al., 2019). Overexpression of this efflux pump not only contributes to resistance but also affects pathogenesis, including enhanced colonization of lung tissue (Rafiei et al., 2022; Yoon et al., 2016). In addition to resistance, the success of Acinetobacter baumannii infections is highly influenced by its ability to acquire nutrients from the host, particularly iron. The lungs contain relatively high iron reserves (0.4–0.9 mg Fe/g dry tissue), which bacteria can exploit to support growth and colonization (Zhang et al., 2019). A critical virulence factor in this mechanism is the production of the siderophore acinetobactin, encoded by the basD gene. This gene is essential for acinetobactin biosynthesis, enabling Acinetobacter baumannii to sequester iron from the host. Mutations or loss of basD have been shown to reduce bacterial colonization and attenuate virulence (Conde-Pérez et al., 2021; Hasan et al., 2015).

To date, the detection of the adeB and basD genes has mostly been performed on pure isolates in vitro, resulting in limited information regarding their presence directly within infected tissues. No studies have evaluated the detection of these two genes in the lung tissue of animals infected with Acinetobacter baumannii in vivo. Therefore, this study introduces novelty by examining the presence of the adeB and basD genes in the lung tissue of infected Rattus norvegicus. This novelty provides new insights into the dynamics of detecting bacterial virulence and resistance genes within a complex biological environment that differs from in vitro conditions. This study aims to detect the presence of adeB and basD in the lung tissues of Rattus norvegicus intravenously injected with Acinetobacter baumannii, providing a comprehensive insight into the roles of these genes as molecular markers of infection.

METHOD

This study was conducted from June to September 2025 at the Molecular Biology Laboratory, Sekolah Tinggi Ilmu Kesehatan Nasional Surakarta. This study was an experimental study aimed at detecting the adeB and basD genes in lung tissues of Rattus norvegicus that were intravenously injected with Acinetobacter baumannii. The study subjects were healthy female Rattus norvegicus aged 2–3 months with normal activity and behavior. Inclusion criteria included animals free from signs of disease or abnormal behavior. A total of six rats were used and divided into treatment groups with observation times of 10 and 18 hours after injection. The injection was carried out using a suspension of clinical isolates of Acinetobacter baumannii that had been prepared in 0.9% physiological NaCl solution at a final concentration of 1×10^7 CFU/ml. Each rat received a single injection of 0.1 ml of bacterial suspension at a concentration of 1×10^7 CFU/ml via the intravenous route (Tayabali et al., 2024).

At 10 hours and 18 hours post-injection, the rats were euthanized using the cervical dislocation method to ensure humane handling. Dissection was also performed on the control group, lung tissues were collected and then washed using 0.9% NaCl to remove residual blood, and approximately 0.4 g of tissue was taken for further analysis (Pérez-Brocal et al., 2020). The lung tissues were then cultured using the streak method on Nutrient Agar (NA) media with a sterile inoculation needle and incubated at 37°C for 24 hours. In addition, part of the tissue was inoculated into Brain Heart Infusion (BHI) broth media and incubated under the same conditions (Anduni et

al., 2024). The BHI culture was subsequently used as material for DNA isolation using the Presto Mini gDNA Bacteria Kit.

DNA amplification was carried out using the Polymerase Chain Reaction (PCR) method to detect the adeB gene using the forward primer 5'-ATGGTTTGGAATGAAGGGTT-3' and reverse primer 5'-AATTGCCCAGCTTTCATAGA-3' (amplicon 549 bp), as well as the basD gene using the forward primer 5'-TGCTCCAGTATTACCGATTG-3' and reverse primer 5'-TAAGTTGAACAGGTGAGAGC-3' (amplicon 533 bp). Both pairs of primers were designed using Primer3Plus. The thermal cycling conditions consisted of initial denaturation at 95°C for 3 minutes, followed by denaturation at 95°C for 30 seconds, annealing at 57°C for 30 seconds, and extension at 72°C for 30 seconds. The PCR products were then analyzed on 1% agarose gel using the same electrophoresis procedure and visualized using a Bio-Rad UV gel documentation system. Data analysis was performed descriptively by observing and interpreting DNA banding patterns to determine the presence of the adeB and basD genes in the lung tissue samples (KEPK/UMP/210/XI/2025).

RESULT

This study was conducted on six lung tissue culture samples, consisting of negative control groups, 10-hour, and 18-hour post-infection groups, as well as one *Acinetobacter baumannii* suspension sample as a positive control. The first stage involved DNA isolation from liquid BHI, followed by PCR testing. DNA isolation was performed on seven samples. DNA isolation was carried out using the Geneaid SYNC™ DNA Extraction tissue sample isolation kit, which involved several process stages, namely sample preparation, cell lysis, DNA binding, washing, and elution. The obtained DNA isolates were then subjected to qualitative testing using electrophoresis. The quality of the isolated DNA was tested by electrophoresis at 90 volts, 400 mAh for 60 minutes, after which the agarose gel was visualized using a Bio UV-Transilluminator.

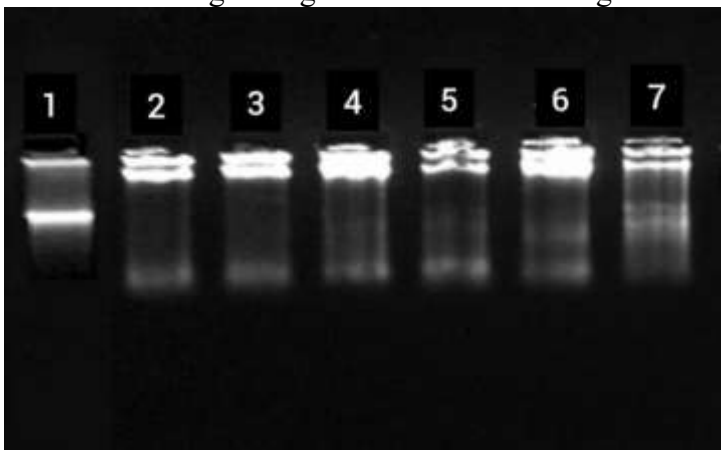

Figure 1. Qualitative DNA test results.
(1) Positive control, (2) Negative control A, (3) Negative control B, (4) 10-hour post-infection A, (5) 10-hour post-infection B, (6) 18-hour post-infection A, (7) 18-hour post-infection B

The qualitative test results showed DNA bands of varying thickness. The thickness or thinness of DNA bands may be caused by differences in DNA concentration.

Table 1.
Quantitative DNA test results using UV-VIS Spectrophotometer

Sample	λ 260	λ 280	DNA Concentration (λ260 × 50 ng/µl × dilution factor)	DNA Purity (λ260/λ280)
1	0,0253	0,0234	253	1,0811
2	0,0297	0,0181	297	1,6408
3	0,0178	0,0209	178	0,8516
4	0,0247	0,075	247	0,3293
5	0,0215	0,0214	215	1,0046
6	0,0266	0,0232	266	1,1465
7	0,0221	0,0208	221	1,0625

* Good DNA purity ranges from 1.8–2.0, and good DNA concentration is above 100 ng/µl

Quantitative testing of DNA isolates showed that the lowest DNA concentration was 178 ng/µl in sample code 3, while the highest DNA concentration was 297 ng/µl in sample code 2. The purity results of the DNA isolates showed that all samples had purity levels below 1.8–2.0, ranging from 0.3293 to 1.6408.

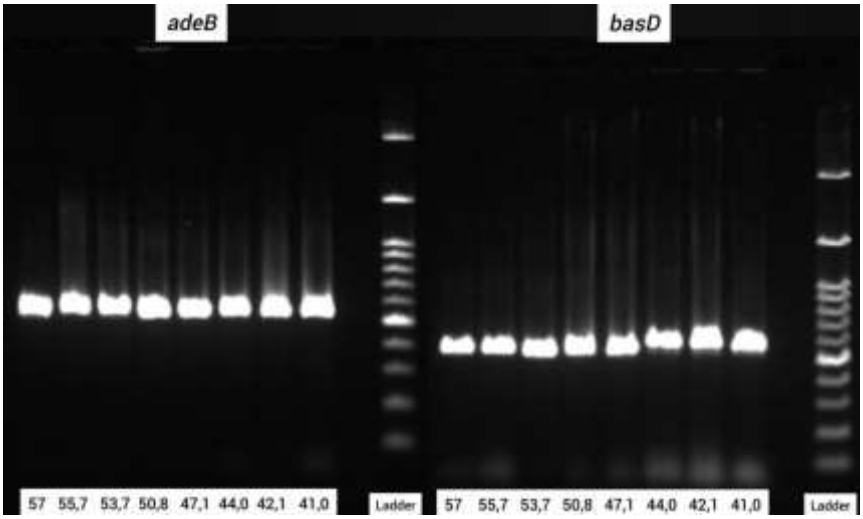

Figure 2. PCR temperature optimization for *adeB* and *basD* genes (Gradient Temperatures 41–57°C)

PCR temperature optimization was performed using the positive control for the *adeB* and *basD* genes. The best optimization results were obtained at 57°C, where clear and distinct DNA bands were observed at lengths of 549 bp for *adeB* and 533 bp for *basD*.

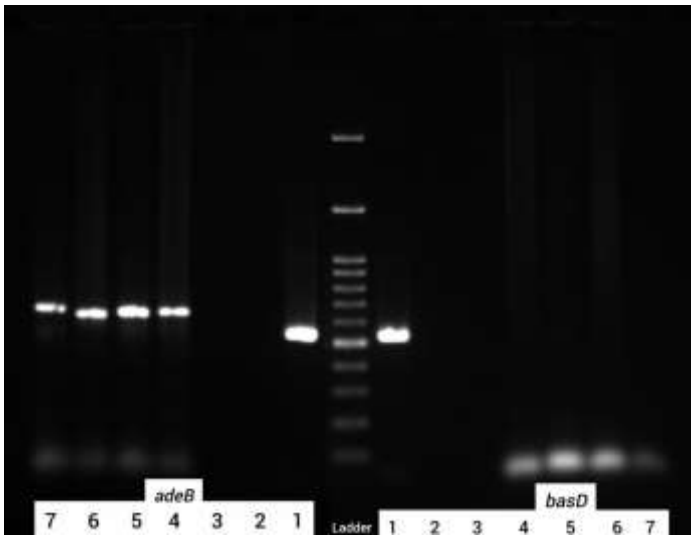

Figure 3. Electrophoresis visualization of PCR results for *adeB* and *basD* genes

PCR detection results of the *adeB* and *basD* genes in rat lung tissue cultures showed the presence of thick and clear DNA bands at number 1, with product lengths of 549 bp for *adeB* and 533 bp for *basD*. However, for numbers 4–7, the *adeB* gene showed thick and clear non-specific DNA bands, and for numbers 2–7, the *basD* gene showed faint non-specific DNA bands.

DISCUSSION

The qualitative DNA test results in Figure 1 show that code (1), the positive control, exhibits a DNA band with clear intensity, although it does not appear full. This condition can occur because DNA electrophoresis only provides a qualitative picture of the presence of fragments, not absolute quantity. Variations in band intensity can also be influenced by the amount of template inserted, polymerase efficiency, and the distribution of DNA fragments during amplification (Wadsworth, 2012). In codes (2) and (3), negative controls A and B, DNA bands are still visible. This can be explained by the nonspecific nature of qualitative tests, which detect only bacterial DNA but can also detect the presence of other DNA in the sample, such as host tissue DNA or extracted biological contaminants. The appearance of a band in the negative control does not indicate specific amplification of the target gene, but rather represents the total DNA that was successfully separated. Previous research has also reported that the presence of host DNA or interfering molecules can cause nonspecific bands in negative controls in PCR-based assays (Jansriphibul et al., 2025).

Thus, the bands in the negative control do not indicate test failure, but rather a consequence of the qualitative method that detects DNA comprehensively, not just the desired gene target. Meanwhile, codes (4) and (5) are groups 10 hours post-infection, which are clearly visualized, indicating that Acinetobacter baumannii is able to colonize and survive in the host lung tissue from the early phase of infection. A similar thing was also observed in the 18-hour post-infection group (codes 6 and 7), where DNA bands remained visible with relatively comparable intensity. This finding indicates that the bacterial load in the lung tissue at both time points was at a level sufficient to be detected by conventional PCR. The absence of a significant difference in intensity between 10 hours and 18 hours can be explained by the bacterial colonization pattern that tends to reach stability in the host tissue. After the initial colonization phase, the dynamics between bacterial growth and the host immune response can balance the population so that DNA accumulation does not increase drastically. This is consistent with research (Ahmad et al., 2019) which mentions the existence of variations in the growth phase and survival mechanisms of Acinetobacter baumannii under in vivo conditions. Furthermore, the limited sensitivity of conventional PCR can mean that small differences in DNA quantity over time are not always visually reflected in the gel bands (Tansho-Nagakawa et al., 2021).

Good-quality DNA has a purity between 1.8 and 2.0 ng/μL, measured using a spectrophotometer (Dewanata & Mushlih, 2021). A high concentration does not necessarily indicate high purity; this can be due to the influence of 1280 or contaminant values. High concentrations can be caused by the presence of RNA molecules capable of absorbing A260, as not only DNA can absorb A260, but RNA can as well (Kusumawati et al., 2023). A low A260 /A280 ratio (≤ ~1.6) can indicate the presence of protein or phenol; and a low A260/A230 ratio can indicate contamination of organic compounds (polysaccharides, polyphenols) in the isolated DNA (Revathy et al., 2025). Furthermore, purity can be affected by the use of unclear cuvettes, which can affect UV light absorbance. Cuvettes that cannot absorb radiation, and technical errors during measurement. Insufficiently homogenized samples, poor sample quality, and improper pipetting can cause DNA to break into small fragments (Dewanata & Mushlih, 2021).

The stages of the Polymerase Chain Reaction (PCR) include denaturation, annealing, and extension. Prior to PCR amplification, annealing temperature optimization is required. The annealing temperature is adjusted during the PCR cycle to allow the primers to bind to the target DNA. This temperature is crucial for ensuring the specificity of primer binding to the desired target. The annealing temperature for each primer pair is determined based on the melting temperature (Tm) specified by the supplier (primer synthesis results) (Aulia et al., 2023). Temperature optimization was performed in the range of 41°C–57°C for both genes, as it follows the calculated Tm temperature of primer synthesis. Figure 2 shows that the primers amplified DNA well and showed the presence of DNA bands corresponding to the 549bp (adeB gene) and 533bp (basD gene) targets. Selection at 57°C was based on the sharp band boundaries, band clarity, and the absence of double bands.

The results of PCR amplification of the adeB gene in this study showed that only the positive control (1) (Acinetobacter baumannii isolate) produced a DNA band at the target size of ±549 bp, consistent with the primer design used. This indicates that the primers used were specific and the PCR conditions applied were valid for detecting the adeB gene. Negative controls (2-3) showed no DNA band, thus confirming the absence of target DNA contamination in the PCR reaction. However, in lung tissue samples from mice intravenously injected with Acinetobacter baumannii 10 and 18hours post-infection (samples 4-7), no DNA band at the 549 bp size was found. These results indicate that the adeB gene was not detected in the lung tissue analyzed.

Several key factors influence bacterial detection in tissue specimens, which we have categorized into three categories: tissue sampling, which includes sampling location and sample quantity; bacterial distribution within the tissue, which involves bacterial load, distribution, and aggregation level; and bacterial detection methods, which involve analytical sample volume and detection method. When bacteria are concentrated in a small, localized location and are not evenly distributed, or are present in low numbers, the probability of detection can be low (Jakobsen et al., 2025). The adeB gene is almost always present in isolates, but its expression can vary significantly depending on conditions. This suggests that even if the gene is present, if the bacteria are few or inactive, detection can be negative (Nageeb et al., 2023). Some inhibitors delay the detection time for the initial amplicon (urea, IgG, bile salts, and calcium chloride), some quench the fluorescence of the DNA-dye complex (hematin and humic acid), and some reduce the final amount of DNA amplicon product formed (hematin, humic acid, and tannic acid) (May Khat New et al., 2024). In mouse lung tissue, it is very possible that blood, ruptured cells (hemolysis), tissue proteins, or other cellular material may be extracted with the bacterial DNA and inhibit PCR. Furthermore, the distribution of bacteria in lung tissue is not always uniform but can be concentrated in certain areas, so tissue samples taken may not include areas with high bacterial populations. This explains why positive controls of bacterial isolates amplified successfully, while more complex tissue samples failed to amplify.

In addition to intact genomic DNA, PCR inhibition can be produced by other forms of DNA, including cDNA or fragmented DNA, such as ancient DNA or DNA extracted from FFPE tissue. Nucleic acid amplification can also be performed using methods other than PCR, such as the halo amplification method. It is possible that if the temperature is too low, the primers can bind to non-target DNA and prevent sensitive detection. Therefore, the interaction of the primers with genomic (non-target) DNA can result in non-specific amplification (mis-priming) (Sue Latham et al., 2023). Research conducted by (Ke Huang et al., 2024) shows that although primers appear "specific," partial similarity or mismatch can reduce sensitivity if the correct polymerase is not selected. Primers are able to bind to DNA sequences that have partial similarity to targets in other bacterial genomes. As a result, non-target bands appear on the electrophoresis gel. This may explain the presence of bands in samples 4-7 that do not correspond to the 549 bp target.

The results of basD gene amplification are shown in Figure 2. The positive control (k+), which contained a pure isolate of Acinetobacter baumannii, showed a DNA band measuring ±533 bp, indicating that the primers and PCR conditions were functioning properly. In contrast, no band was found in the negative controls (ka and kb), confirming the absence of contamination. In mouse lung tissue samples taken 10 and 18hours post-infection (codes 10a, 10b, 18a, and 18b), no DNA band representing the basD gene was observed, in contrast to the results from the pure bacterial control. These results indicate that the basD gene was not detected in the analyzed lung tissue. BasD gene expression in Acinetobacter baumannii is an essential part of the acinetobactin siderophore system, which functions in the acquisition of iron, an essential nutrient for pathogenic bacteria. This gene is regulated by the Ferric Uptake Regulator (Fur), which is sensitive to iron availability in the host environment. Under iron-depleted conditions, Fur is inactivated, and siderophore gene transcription is induced to enable acinetobactin production. However, under iron-rich conditions, for example in lung tissue that may contain hemoglobin, transferrin, or ferritin, Fur is active and suppresses basD expression (Kim et al., 2021).

This suppression represents an energy-saving strategy, as the bacteria do not need to activate additional acquisition mechanisms if sufficient nutrients are available from the host. Interestingly, despite repressing basD expression, Acinetobacter baumannii remains able to colonize by utilizing alternative virulence factors. One of these is outer membrane proteins (OMPs), which play a role in adhesion to host cells and interaction with extracellular matrix components. Research (Reyes et al., 2022) shows that adhesion via OMPs can serve as an effective colonization strategy, even when the siderophore system is not dominant. Therefore, the absence of basD detection in lung tissue does not necessarily indicate a loss of colonization ability, but rather reflects the pathogen's flexibility in selecting virulence strategies according to the host's environmental conditions. DNA extraction from animal tissues often leaves PCR-inhibiting molecules, including hemoglobin, lipids, and cellular metabolites. These compounds can interact with the polymerase enzyme and reduce amplification efficiency, thus decreasing the sensitivity of the PCR method (Tansho-Nagakawa et al., 2021). This explains the difference in results between pure bacterial controls, which are relatively free of inhibitors, and lung tissue samples loaded with endogenous components.

Furthermore, the inflammatory state of infected lung tissue also increases the activity of host enzymes and immune cells, making bacterial DNA more susceptible to degradation. (Suresh et al., 2019) reported that host nuclease activity plays a significant role in pathogen DNA degradation in tissue, which can reduce the success of molecular detection. Similar findings were reported by (Delaplace et al., 2021), who showed that DNA degradation in inflamed tissue makes it difficult to amplify target genes, even though they are still present in small amounts. This could therefore explain why genes cannot be visualized in DNA isolates extracted from lung tissue.

CONCLUSION

This study shows that the adeB and basD genes can be detected in pure Acinetobacter baumannii isolates but not in mouse lung tissue 10 and 18 hours post-infection. These results indicate that the presence of target genes in host tissue cannot always be identified using conventional PCR due to the influence of host biology, bacterial load, and method limitations. These findings emphasize the need for more sensitive detection approaches and consideration of host factors in the analysis of pathogenic bacterial gene expression.

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