Antibiotic resistance assessment of *Acinetobacter baumannii* isolates from Tehran hospitals due to the presence of efflux pumps encoding genes (*adeA* and *adeS* genes) by molecular method

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**Abstract**

**Objective:** *Acinetobacter baumannii* (*A. baumannii*) has caused many problems in nosocomial infections. Efflux pumps are considered as one of the most important mechanisms of resistance in this bacterium and have the ability to excrete toxic substances such as antibiotics out of the cell.

**Results:** In this study, 60 isolates of *A. baumannii* were collected from patients in several hospitals in Tehran, Iran. After diagnosis using standard biochemical methods, the pattern of antibiotic susceptibility was determined using the disk diffusion method according to CLSI guidelines. The *adeA* and *adeS* genes were identified by PCR method. The highest resistance to Piperacillin and the lowest resistance to Gentamicin were observed (100% compared to 48.4%). 6.6% of the isolates had only *adeA* gene and *adeS* gene was observed in 8.4% of isolates and both genes were detected in 73.4% of the samples. Despite the high resistance of *A. baumannii* to antibiotics and due to the high frequency of genes of *adeA* and *adeS* efflux pumps in *A. baumannii* isolates, it can be concluded that these efflux pumps may play an important role in resistance of this bacterium. By determining the pattern of antibiotic the resistance before treatment, the resistance of this pathogen can be prevented in societies.

**Keywords:** *Acinetobacter baumannii*, Efflux pump, *adeA* gene, *adeS* gene, Antibiotic resistance

**Introduction**

Nosocomial infections are known as one of the crucial problems for public health [1]. Most of these infections are caused by Gram-negative bacilli [2]. One of the most important causes of nosocomial infections is *A. baumannii* that is Gram-negative Coccobacilli and aerobic and prefer humid environments for a living [3, 4]. The most important pathogen of this genus is, *A. baumannii* which can cause a wide range of diseases and nosocomial infections such as pneumonia, septicemia, urinary tract infections, skin and wound infections, endocarditis, and meningitis [5, 6]. Infection with this bacterium is increasing in people with immunodeficiency or cancer [7]. One of the main problems caused by this bacterium is the development of high antibiotic resistance [8]. One of the mechanisms that this bacterium uses to resist various antibiotics is the use of efflux pumps. By using efflux pumps, the *A. baumannii* can direct antibiotics outwards and prevent antibiotics from affecting the bacteria [9].
Due to the high prevalence of efflux pump genes especially AdeA and AdeB that are the major discovered efflux pumps in *A. baumannii*, this may play a significant role in the antibiotic resistance of *A. baumannii* isolates. Identifying the antibiotic susceptibility pattern is essential to prevent the prevalence of antibiotic resistance. For this purpose, this study was performed by determining the antibiotic susceptibility in clinical trials of *A. baumannii* collected from several hospitals in Tehran.

### Main text

#### Materials and methods

**Patients and sampling**

In this study, 60 *A. baumannii* isolates were collected from 100 different samples including chips, blood, urine, wound culture, respiratory secretions, catheters, spinal fluid, and pleurisy during the 9 months from August 2018 to May 2019 from different departments of Milad Hospital, Baqiyatallah and the Rasool-Akram hospital of Tehran was identified.

1. Isolation and identification of *A. baumannii* strains

   Prevalent biochemical tests and methods have been used to identify and confirm *A. baumannii* strains [10]. The specimens were inoculated on blood agar (Merck) and MacConkey agar (Merck) medium and incubated for 24 h at 37 °C. Conventional biochemical methods such as oxidase, citrate, urea urease, malonate consumption, oxidation and fermentation of sugars, motility, and indole production were done to identify *A. baumannii* [11].

2. Confirmation of *Acinetobacter baumannii* by PCR

   To confirm the *Acinetobacter baumannii*, the gene *bla* OXA-51-like was examined by PCR similar to the method performed by Jia et al. [12]. Because it has been made clear that, there is OXA-51–like gene in *Acinetobacter baumannii* isolates instinctively [13].

**Antimicrobial susceptibility test**

This test was performed using the Kirby Bauer method using 9 disks including Imipenem (10 μg), meropenem (10 μg), Gentamicin, Pipercillin, Ampicillin-Sulbactam, Ceftazidime, Amikacin, Tetracycline, and Ciprofloxacin. The 2019 CLSI was interpreted [14]. The *Acinetobacter baumannii* ATCC19606 was used as quality control for this test.

**DNA extraction**

DNA extracting was performed by DNA extraction kit (Bioneer Company Korea, Cat. No. K-3032-2-) was used.

**Identification of adeA and adeS gene via PCR**

PCR was performed to screen for adeA and adeS genes. The primer sequences used are shown in Table 1. The PCR program For amplification was: an initial denaturing step, 5 min at 94 °C, 30 cycles of the 30 s at 94 °C, primer connection for adeA at 55.5 °C and For adeS at 54.5 °C for the 30 s, 90 s at 72 °C, and 5 min as a final extension at 72 °C were performed. The PCR products were analyzed through a 2% agarose gel containing Syber safe and were detected using a gel documentary device.

The primers designed in this study are as follows: VIM-1  5′-TGGTTGTATACGTCCCGTCA TGTGTG CTGGAGCAAGTCTA-3′, IMP-15′-TAACGGGTGGGG CGTTGTCTCC CGCCCGTGTGCTGATGAA-3′ and The third primer for OXA-51 was described previously [11].

Primers for adeA and adeS genes:

- **adeA** Forward 5′- TTGATCGTGCTTCTATTCCTC
- **adeA** Reverse 5′-GGCTGCACCCTAGTATTCGTT-3′ [15]
- **adeS** Forward 5′-TGCCGCAAATTCCTATTCC-3′
- **adeS** Reverse 5′-TTAGTCACGGCGACCTCTCT-3′ [16].

**Statistical analysis**

Questionnaire information and the results of phenotypic and genotypic experiments were analyzed using SPSS software, version 23, by the Chai test and Fisher’s exact test.

**Results**

In this study, 60 bacterial isolates were collected as *Acinetobacter baumannii* from three hospitals in Tehran. The frequency distribution of *Acinetobacter baumannii* isolates according to the sample is shown in Table 1.

### Table 1 Frequency distribution of *Acinetobacter baumannii* isolated from patients according to the type of clinical sample and hospital

| Sample type   | Baqiyatallah | Rasool-Akram | Milad N (%) |
|---------------|--------------|--------------|-------------|
| Tracheal      | 2            | 1            | 1           | 4 (6.6) |
| Wound         | 6            | 3            | 4           | 13 (21.8) |
| Urine         | 3            | 2            | 2           | 7 (11.8) |
| Blood         | 6            | 5            | 5           | 16 (26.6) |
| Catheter      | 1            | 1            | 2           | 4 (6.6) |
|               | 7            | 3            | 6           | 16 (26.6) |

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[10] Jia et al., 12. [11] Jia et al., 13. [12] Jia et al., 14. [15] Jia et al., 16. [16] Jia et al.
Results of genotypic tests
The presence of the OXA-51 gene in the Acinetobacter baumannii was investigated. All samples in the PCR method had this gene. The frequency distribution of the adeA and adeS genes in the PCR method was also examined. The frequency of adeA and adeS genes in the samples were 6.6% and 8.4%, respectively calculated by PCR method. In 44 (73.4%) of the samples, these two genes were present together and in 7 (11.6%) of the samples, none of the adeA and adeS genes were present.

Results of the relationship between antibiotic resistance and efflux pump genes
According to the results, it can be concluded that the expression of adeA and adeS genes are related to the Tetracycline, Ciprofloxacin, Gentamicin, and Amikacin resistance (Table 3).

Studies show that in addition to efflux adeABC pumps, other efflux pumps also have a leading role to play in developing resistance to these antibiotics, and the simultaneous presence of these efflux pumps increases resistance. Although other factors contribute to this antibiotic resistance, efflux pumps play the most crucial role in creating resistance to these antibiotics [17].

Discussion
Nosocomial infections have become one of the main problems in treatment. The most common cause of nosocomial infections is Gram-negative bacteria. Acinetobacter baumannii is an opportunistic hospital pathogen that causes a wide range of nosocomial infections. Due to the indiscriminate use of broad-spectrum antibiotics by people, we are witnessing high antibiotic resistance caused by this bacterium. The high antibiotic resistance of this bacterium is associated with the proliferation of multiple antibiotic resistance genes. Various studies have shown that Acinetobacter baumannii is resistant to most Beta-lactam antibiotics and Quinolones, and its resistance to Aminoglycosides is increasing. In this study, the most effective antibiotics used against Acinetobacter baumannii were Gentamicin and Imipenem, which had 48.4% and 50% resistance, respectively, while the highest resistance was to Piperacillin (100%), Ceftazidime (98.4%), and Amikacin (96.6%) and Tetracycline (91.6%).

In the present study, the resistance of Acinetobacter baumannii to at least one antibiotic was seen in three groups or more in all isolates. Therefore, the frequency of multi-drug resistance in Acinetobacter baumannii isolates was 100%, but the frequency of Acinetobacter baumannii with multiple resistance in other studies reported was reported to be 50 to 100% variable [12]. High MDR levels in Acinetobacter baumannii studied in Farsiani et al. Studies in Iran and Rynga et al. In India were reported to be 97% and 85%, respectively [18, 19], which could be due to abuse of antibiotics.

In the Noori et al. study, All isolates had 100% resistance to Ceftazidime, Ciprofloxacin, and Piperacillin, and the frequency of the adeS gene was 91% which in comparison to Ciprofloxacin adeS resistance was different [20]. According to the results of the present study, we saw a decrease in antibiotic resistance to ceftazidime and ciprofloxacin and the same resistance to piperacillin was reported.

Research by Rahbarnia et al. Found that the resistance to Ciprofloxacin was 95%, Imipenem 82%, and Gentamicin 35%. Also, the prevalence of MDR and XDR

### Table 2 Antibiotic resistance pattern of Acinetobacter baumannii isolates

| Antibiotic          | Resistant N (%) | Intermediate N (%) | Susceptible N (%) |
|---------------------|-----------------|--------------------|-------------------|
| Piperacillin        | 60 (100)        | 0 (0)              | 0 (0)             |
| Ceftazidime        | 59 (98.4)       | 1 (1.6)            | 0 (0)             |
| Amikacin           | 58 (96.6)       | 0 (0)              | 2 (3.4)           |
| Tetracycline       | 55 (91.6)       | 2 (3.4)            | 3 (5)             |
| Ampicillin-Sulbactame | 39 (65.0)    | 2 (3.4)            | 19 (31.6)         |
| Meropenem          | 38 (63.4)       | 1 (1.6)            | 21 (35)           |
| Ciprofloxacin      | 37 (61.6)       | 3 (5.0)            | 20 (33.4)         |
| Imipenem           | 30 (50.0)       | 0 (0)              | 30 (50.0)         |
| Gentamicin         | 29 (48.4)       | 0 (0)              | 31 (21.6)         |

### Table 3 Antibiotic resistance in acinetobacter bacterial isolates

| Antibiotic | Resistant N (%) | Resistant. adeA gene (%) | Resistant. adeS gene (%) |
|------------|-----------------|--------------------------|--------------------------|
| Amikacin   | 58 (96.6)       | 48                        | 49                       |
| Tetracycline | 55 (91.6)   | 36                        | 36                       |
| Ciprofloxacin | 37 (61.6) | 29                        | 29                       |
| Gentamicin | 29 (48.4)       | 48                        | 49                       |
in the studied strains was 76% and 30%, respectively, which compared to the present study, higher resistance to Ciprofloxacin and Imipenem has been reported [21]. This reason for the increase in antibiotic resistance may be related to different geographical areas in Iran.

In Abdar et al. study, the resistance to Meropenem and Ceftazidime was reported to be 71% and 93%, respectively, which is very similar to the present study [22].

According to Fallah et al., The antibiotic resistance of Acinetobacter baumannii isolates is as follows: 95.4% to Ceftazidime, 91.7% to Meropenem, and 92.6% to Ciprofloxacin, which is reported to have a higher resistance to Meropenem and Ciprofloxacin than the present study [23]. The reason for the decrease in antibiotic resistance in recent years in Iran can be due to changes in antibiotic treatment policies and the use of antibiograms before prescribing antibiotics.

According to a report by Al-Agamy et al., 100% of Acinetobacter baumannii isolates were 85% resistant to Ciprofloxacin and 70% to Imipenem [24]. Compared to the present study, we saw a decrease in resistance to both antibiotics.

According to the study of Angoti et al., The resistance to Ciprofloxacin is 99%, Ceftazidime, Meropenem, and Imipenem 98%, Gentamicin 77%, Amikacin 48% which is different from the present study and also the prevalence of adeA gene in 61 samples 88.5% was reported [25]. Based on the results of a recent study, we have seen a decrease in antibiotic resistance in recent years in the treatment of patients.

In a study by Boral et al. [26], antibiotic resistance for Ciprofloxacin, Imipenem, Ampicillin/sulbactam, Cef-tazidime, and Amikacin was observed to be 100%, 99.4%, 99.4%, 99.4%, and 91.8%, respectively. That is an increase in reported antibiotic resistance compared to the present study.

In a study by Ranjbar et al., The antibiotic resistance to Ceftazidime, Ciprofloxacin, Piperacillin, Gentamicin, Amikacin, and Ampicillin/Sulbactam was reported to be 75.95%, 96.3%, 95.1%, 92.1%, 87.2%, and 76.1%, respectively [27]. One of the reasons for the difference in antibiotic resistance compared to the recent study is the diverse geographical areas for sampling patients.

In a study by Jia et al. China, resistance to Piperacillin and Ceftazidime was reported 92.2% that was similar to this study [12].

The frequency of Tetracycline resistance in the studied isolates was 91.6%. The results of studies conducted in Iran were similar to the results of studies of Sarhaddi et al. [28] that were 96.3% and compared to the study of Nemec et al. Antibiotics were 62% higher [29].

One of the resistance mechanisms in the Acinetobacter baumannii is the presence of efflux pumps. These pumps cause the leakage of antibiotics and a wide range of substances out of the bacteria, creating multidrug resistance. Three systems, AdeFGH: RND, AdeIJK, and AdeABC, have been identified in the Acinetobacter baumannii, among which AdeABC is most involved in the MDR Acinetobacter baumannii [30]. Although high levels of resistance do not occur only as a result of multi-drug efflux pumps, the expression of their genes among isolates with high antibiotic resistance cannot be ignored. Therefore, it is necessary to identify resistance systems, including efflux pumps.

In the tracking section of efflux pumps encoding genes, 80% (48 samples) of Acinetobacter baumannii isolates had adeA gene and 81.66% (49 samples) isolates had adeS gene, which compared to other studies conducted in Iran, Japanese race et al. [27] (adeA 100%), Beheshti et al. [31] (for all gene pumps AdeABC 100%) and Ardabili et al. [32] (adeS 100%) were lower.

Compared to studies conducted abroad by Jia et al. In China [12], 79.6% of the Acinetobacter baumannii isolates had the adeA gene and 80.6% had the adeS gene, also in the study conducted by Nemec et al. in France [29] 81.9% of the isolates had adeA and adeS genes that were similar to the results of our study but compared to the research by Kor et al. [33] that the frequency of adeA gene in isolates was 62.7% that was higher. The reason for the differences in different studies could be due to differences in the patterns of antibiotic use, the type of clinical sample, the number of samples studied, sampling method, environmental factors and the different geographical distribution of these genes.

Conclusion

A significant increase in antibiotic resistance is one of the main problems in the treatment of infections caused by Acinetobacter baumannii. Efflux pumps play a key role in the development of multiple resistance to antimicrobial drugs. It is important to evaluate the presence of efflux pump genes in preventing the spread of antibiotic resistance and to suggest an appropriate treatment model for patients infected with this bacterium. So the high prevalence of genes encoding efflux pumps in this bacterium is one of the important factors in the spread of antibiotic resistance between isolates in different geographical regions. Of course, the role of other factors and mechanisms involved in the development of Acinetobacter baumannii resistance should not be ignored.
Limitations
Our limitation in this research project was the lack of sufficient funding. If there is more budget, it can be further developed and more methods can be added.

Abbreviations
PCR: Polymerase chain reaction; CLSI: Clinical and Laboratory Standards Institute; MDR: Multi drug resistance; ATCC: American type culture collection; RND: Resistance-nodulation-division; IMP: The genes imipenemase; VIM: Verona integron-encoded metallo-beta-lactamases.

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Authors’ contributions
BBT, MN, MK: design of study. BBT, MK, HA, FS, SB: evaluation of data, preparation of the manuscript. MK, HA, FS, SB: assessment of data. All authors read and approved the final manuscript.

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Availability of data and materials
All relevant data are included in the manuscript.

Ethics approval and consent to participate
This study was in accordance with the declaration of Helsinki. This study was approved by the Ethics Committee of Shahed University of Medical Sciences. The informed consent was obtained from all the participants, and informed consent obtained was written.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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