

# Molecular survey of aminoglycoside-resistant *Acinetobacter baumannii* isolated from tertiary hospitals in Qazvin, Iran

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## Abstract

Aminoglycoside-modifying enzymes (AMEs) and 16S rRNA methylases (16S RMTase) are two main resistance mechanisms against aminoglycosides. This study aimed to evaluate the frequency of AMEs and 16S rRNA methylase genes among aminoglycoside non-susceptible *Acinetobacter baumannii* isolates and to assess their clonal relationship using repetitive extragenic palindromic-PCR (rep-PCR). In this cross-sectional study, a total of 192 *A. baumannii* isolates were collected from the patients hospitalized in Qazvin, Iran (January 2016 to January 2018). Identification of isolates was performed by standard laboratory methods and API 20E strips. Antimicrobial susceptibility was determined by Kirby–Bauer method followed by examination of the genes encoding the AMEs and 16S RMTase by PCR and sequencing methods. The clonal relationship of isolates was carried out by rep-PCR. In total, 98.4% of isolates were non-susceptible to aminoglycosides, 98.4%, 97.9% and 83.9% of isolates were found to be non-susceptible against gentamicin, tobramycin and amikacin, respectively. The frequencies of *aph(3')-VI*, *aac(6')-Ib*, *aac(3)-II*, *aph(3')-Ia* and *armA* genes were 59.3%, 39.2%, 39.2%, 31.7% and 69.8%, respectively, either alone or in combination. Rep-PCR results showed that the aminoglycoside non-susceptible isolates belonged to three distinct clones: A (79.4%), B (17.5%) and C (3.2%). The findings of this study showed a high frequency for AMEs with the emergence of *armA* genes among the aminoglycoside non-susceptible *A. baumannii* isolates. Rational administration of aminoglycosides as well as using an appropriate infection control policy may reduce the presence of resistance to antibiotics in medical centres.

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**Keywords:** 16S rRNA methylases, *Acinetobacter baumannii*, aminoglycoside-modifying enzymes, repetitive extragenic palindromic-PCR

**Original Submission:** 15 October 2020; **Revised Submission:** 10 April 2021; **Accepted:** 19 April 2021

**Article published online:** 23 April 2021

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## Introduction

*Acinetobacter baumannii* is a clinically important Gram-negative pathogen in medical centres. This bacterium is responsible for various types of nosocomial infections including pneumonia, bacteraemia, surgical site infections, and urinary tract infections [1]. In recent years, the emergence of multidrug resistance to antibiotics has become a major clinical concern for physicians.

This problem leads to serious limitations in the treatment of patients infected with these pathogens, and to increased morbidity and mortality [2,3]. Aminoglycosides are the most frequently used antibiotic agents among topically applied antibiotics in the treatment of infections caused by Gram-negative bacteria. Combining an aminoglycoside with a  $\beta$ -lactam is considered to be more effective treatment against infections caused by Gram-negative bacteria [4]. These antibiotics block protein synthesis in the bacterium by binding to 30S ribosome and eventually lead to bacterial death [5]. Indiscriminate use of these antibiotics increases antibiotic resistance in bacteria and makes the therapy ineffective [6]. Resistance to aminoglycosides may occur based on the following mechanisms: (a) drug inactivation using aminoglycoside-modifying enzymes (AMEs), (b) ribosomal binding site alterations, (c) reduction of antibiotic enzyme regulation by down-regulation of porin genes; and (d)