



ORIGINAL ARTICLE

In vitro activity of aminoglycosides against clinical isolates of *Acinetobacter baumannii* complex and other nonfermentative Gram-negative bacilli causing healthcare-associated bloodstream infections in Taiwan



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testing;

Abstract *Background/Purpose:* Aminoglycosides possess *in vitro* activity against aerobic and facultative Gram-negative bacilli. However, nationwide surveillance on susceptibility data of *Acinetobacter baumannii* complex and *Pseudomonas aeruginosa* to aminoglycosides was limited, and aminoglycoside resistance has emerged in the past decade. We study the *in vitro* susceptibility of *A. baumannii* complex and other nonfermentative Gram-negative bacilli (NFGNB) to aminoglycosides.

Methods: A total of 378 NFGNB blood isolates causing healthcare-associated bloodstream infections during 2008 and 2013 at four medical centers in Taiwan were tested for their susceptibilities to four aminoglycosides using the agar dilution method (gentamicin, amikacin, tobramycin, and isepamicin) and disc diffusion method (isepamicin).

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nonfermentative
Gram-negative
bacilli

Results: *A. baumannii* was highly resistant to all four aminoglycosides (range of susceptibility, 0–4%), whereas >80% of *Acinetobacter nosocomialis* and *Acinetobacter pittii* blood isolates were susceptible to amikacin (susceptibility: 96% and 91%, respectively), tobramycin (susceptibility: 92% and 80%, respectively), and isepamicin (susceptibility: 96% and 80%, respectively). All aminoglycosides except gentamicin possessed good *in vitro* activity (>94%) against *P. aeruginosa*. Amikacin has the best *in vitro* activity against *P. aeruginosa* (susceptibility, 98%), followed by *A. nosocomialis* (96%), and *A. pittii* (91%), whereas tobramycin and isepamicin were less potent against *A. pittii* (both 80%). Aminoglycoside resistances were prevalent in *Stenotrophomonas maltophilia* and *Burkholderia cepacia* complex blood isolates in Taiwan.

Conclusion: Genospecies among the *A. baumannii* complex had heterogeneous susceptibility profiles to aminoglycosides. Aminoglycosides, except gentamicin, remained good *in vitro* antimicrobial activity against *P. aeruginosa*. Further *in vivo* clinical data and continuous resistance monitoring are warranted for clinical practice guidance.

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Introduction

Nonfermentative Gram-negative bacilli (NFGNB) are the leading pathogens that cause nosocomial bacteremia and infections.¹ They result in high fatality in critically ill patients and in patients with septic shock and bacteremia. The mortality rate attributed to NFGNB bacteremia could be as high as 25%.^{2,3} Among NFGNB, *Pseudomonas aeruginosa*, *Acinetobacter baumannii* complex, and *Stenotrophomonas maltophilia* were the most common pathogens causing healthcare-associated bloodstream infections.³

NFGNB are easily resistant to most of the beta-lactam antibiotics through beta-lactamase production, impermeability, and multidrug efflux pumps.⁴ The emerging resistance of NFGNB to all commonly used antibiotics may lead to inappropriate administration of empirical antibiotics, which contributes to the high fatality rate of NFGNB bacteremia.^{2,5,6} The limitation of the susceptible antibiotic spectrum also makes the clinical treatment more difficult. Therefore, periodic active surveillance for the epidemiology of NFGNB resistance is crucial for infection control and antibiotic stewardship.

Aminoglycosides possess *in vitro* activity against many aerobic and facultative Gram-negative bacilli, like *P. aeruginosa* and *Acinetobacter* spp., and they are intrinsically inactive against some NFGNB, such as *S. maltophilia* and *Burkholderia cepacia* complex. Therefore, the aminoglycosides are the drug of choice for antimicrobial combination for severe infections (e.g., bacteremia) caused by NFGNB.⁷ However, resistance to aminoglycosides in *P. aeruginosa* and *Acinetobacter* spp. can develop by enzymatic modification, impermeability, or MexXY (also referred to as *AmrAB*) efflux pumps.^{6,8} The susceptibility data of NFGNB to aminoglycosides were limited in Taiwan, and the previous study did not differentiate *A. baumannii* complex into the species level for investigation.⁹ In this study, we aimed to compare the *in vitro* susceptibility of NFGNB blood isolates to aminoglycosides in patients with healthcare-associated bacteremia in Taiwan.

Methods

Bacterial isolates

Healthcare-associated bloodstream infections were defined as bacteremia occurring in patients admitted over 48 hours, in patients with recent hospitalization history within 90 days, in patients who were nursing home residents, and in patients who were frequent visitors of the hemodialysis facility or the hospital-based clinic. Blood isolates of NFGNB collected during 2008 to 2013 and preserved at four medical centers in Taiwan were retrospectively recruited using their collection numbers from the laboratory. The four hospitals included National Taiwan University Hospital (NTUH) and Taipei Veterans General Hospital (TVGH) located in northern Taiwan; China Medical University Hospital (CMUH) located in central Taiwan; and Kaohsiung Chang Gung Memorial Hospital (KCGMH) located in southern Taiwan. A total of 378 available NFGNB isolates (NTUH, 319 isolates; TVGH, 18 isolates; CMUH, 32 isolates; and KCGMH, 9 isolates) retrospectively identified from 2013 isolates were randomly selected by the collection numbers for aminoglycoside susceptibility testing. Those isolates included *P. aeruginosa* (100 isolates), *A. baumannii* (76 isolates), *Acinetobacter nosocomialis* (54 isolates), *Acinetobacter pittii* (45 isolates), *S. maltophilia* (50 isolates), and *B. cepacia* complex (53 isolates). None of the patients had duplicate bacterial isolates in this study.

Bacterial identification

The species of all isolates, including *A. baumannii* complex strains, were routinely identified using standard conventional microbiological methods or by the Vitek System (bioMérieux, Hazelwood, MO, USA) as required. The genospecies of *A. baumannii* strains were further identified according to the sequence of the 16S-23S rRNA gene intergenic spacer region as previously described.¹⁰

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