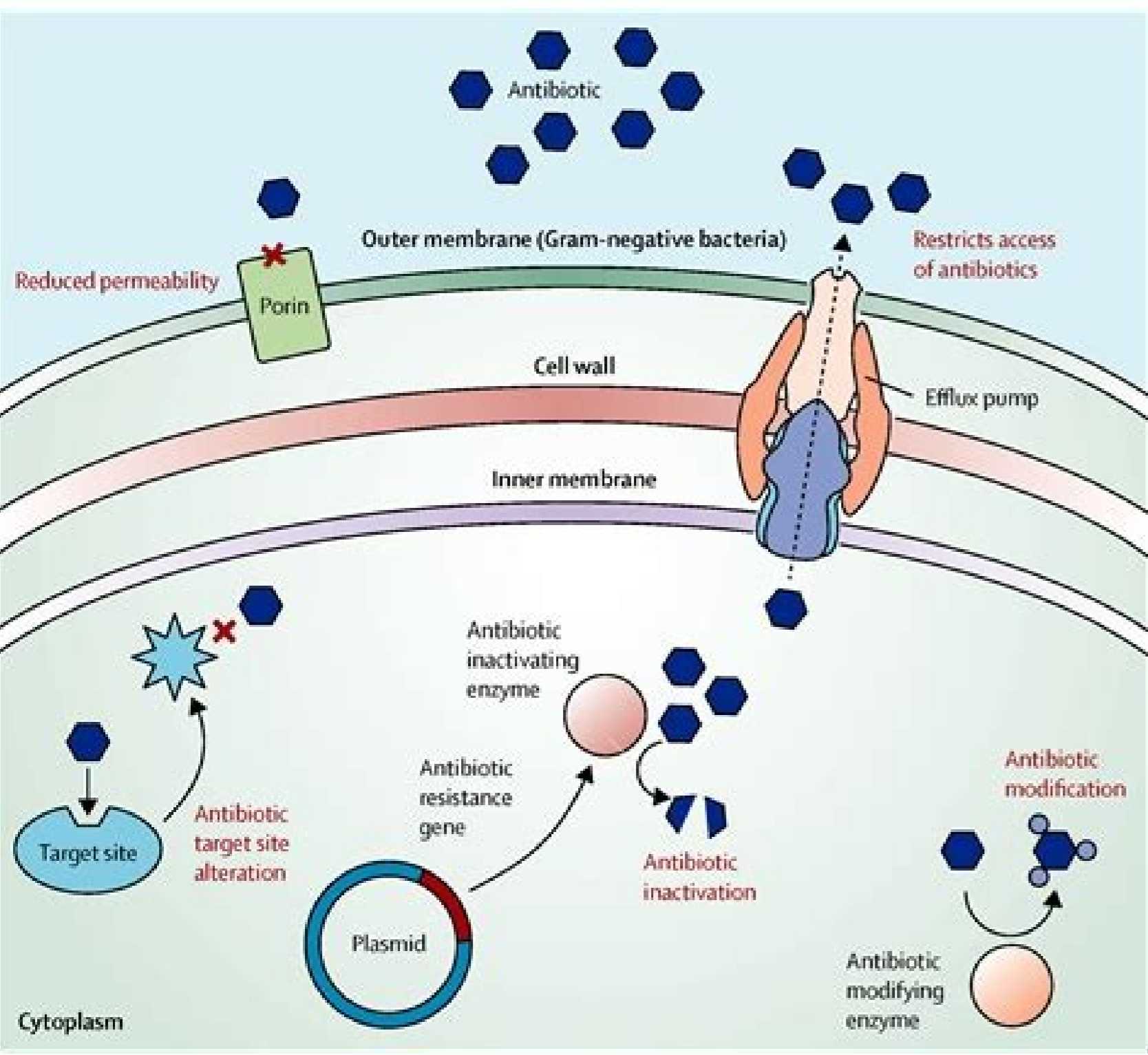




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ANTIBIOTIKOOPORNOSTI IZMENLIVOSTI VITAMINOV PSEUDOMONAS AERUGINOSA S PAMONJI OPOSOBNOSTIJO I GORETJE PSEUDOMONAS

Abstract The aim of the study was to determine the antibiotic resistance and vitamin levels in Pseudomonas aeruginosa strains with different degrees of antibiotic resistance. The study was conducted in the Department of Medical Microbiology, Sakarya University, Turkey. The results showed that the antibiotic resistance and vitamin levels were significantly higher in the multiple drug resistant (MDR) group compared to the other groups. The study also showed that the antibiotic resistance and vitamin levels were significantly higher in the MDR group compared to the other groups.

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ORIGINAL PAPER

The effects of active efflux pumps on antibiotic resistance in Pseudomonas aeruginosa

Huseyin Agah Terzi · Canan Kulah · Ihsan Hakkı Ciftci

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Abstract In this study, we investigated the roles of active efflux pumps in antibiotic resistance. The transcription efflux pump genes were analyzed by real-time polymerase chain reaction (qPCR) to determine their role in drug resistance. Antibiotic sensitivity testing was carried out using the Vitek 2 automated system (bioMérieux, France). Isolates were divided into four groups according to their resistance status: multiple-drug resistant (MDR), isolated carbapenem resistant (ICR), isolated quinolone resistant (IQR), and carbapenem and quinolone resistant (CQR). Transcript levels of *mexB*, *mexD*, *mexF*, and *mexY* were analyzed by qPCR using a LightCycler instrument (Roche, Germany). The genetic similarity between isolates was determined using arbitrarily primed PCR (AP-PCR). Among the 50 isolates investigated, the frequency of genes classified as overexpressed were 88 % for *mexD*, 76 % for *mexB*, 46 % for *mexF*, and 40 % for *mexY*. Within the MDR group, *mexB* was overexpressed in 15 of 22 isolates, *mexD* in 20 of 22, *mexF* in 15 of 22, and *mexY* in 19 of 22. In the ICR group, isolates *mexB* and *mexD* were each overexpressed in five isolates. *mexD* overexpression was observed in all seven CQR isolates. Within the IQR group, *mexB* and *mexD* were overexpressed in all 12 isolates. *mexF* overexpression was detected in 7 of 12 isolates in this group. 18 distinct banding patterns were determined by AP-PCR. Increased transcription of *mexB* was directly correlated with meropenem resistance in the majority of

isolates tested, while *MexCD-OprJ* and *MexEF-OprN* were related to quinolone resistance; the *MexCD-OprJ* efflux pump was also related to multidrug resistance. Increased transcription of *mexY* may contribute to the gentamicin resistance.

Keywords *Pseudomonas aeruginosa* · Resistance mechanism · Efflux pumps · qPCR · AP-PCR

Introduction

The rise of antibiotic resistance is an increasingly important threat, particularly for infections caused by *Pseudomonas aeruginosa*. One of the primary mechanisms driving this resistance is the overexpression of efflux pump systems, which enable resistance to a wide range of drugs with different constitutional features. (Strateva and Yordanov 2009)

The RND family of efflux pumps, including MexAB-OprM, MexCD-OprJ, MexEF-OprN, and MexXY-OprM, represent an important set of efflux systems in *P. aeruginosa*, with a broad range of drug specificities. MexAB-OprM was the first efflux pump found to target multiple classes of drugs, including fluoroquinolones, tetracyclines, chloramphenicol, β -lactams and β -lactamase inhibitors, macrolides, novobiocin, trimethoprim, and sulfonamides. (Köhler et al. 1996; Li et al. 1995, 1998). MexCD-OprJ exhibits a high degree of sequence similarity to MexAB-OprM, and has also been shown to extrude a variety of antimicrobial agents, including fluoroquinolones, β -lactams, chloramphenicol, tetracycline, novobiocin, trimethoprim, and macrolides (Poole et al. 1996). Other efflux pumps within this family exhibit more narrow spectra of activity; β -lactams are poor substrates for MexCD-OprJ

H. A. Terzi (✉) · I. H. Ciftci
 Department of Medical Microbiology, Sakarya University
 Training and Research Hospital, Sakarya, Turkey
 e-mail: agah.terzi@yahoo.com

C. Kulah
 Department of Medical Microbiology, Bilecik Ecevit University,
 School of Medicine, Zonguldak, Turkey



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RESEARCH ARTICLE
 Open Access

Pseudomonas aeruginosa β -lactamase induction requires two permeases, AmpG and AmpP

Kiki-Fa Kang¹, Alan Aguilu¹, Lisa Schepet¹, Katali Mathai²

Abstract
Background In Enterobacteriaceae, β -lactam antibiotic resistance involves multiple recycling intermediates. Murin recycling is a complex process with discrete steps taking place in the periplasm and the cytoplasm. The AmpG permease is critical to this process as it transports hydrolyzed antibiotic fragments across the inner membrane in Pseudomonas. The intrinsic mechanism remains to be elucidated. Since the mechanism involves two crucial components, the characterization of transporters is vital to establish the link.
Results *Pseudomonas aeruginosa* PAO1 has two amp genes, *AmpG1* (*ampG*) and *AmpG2* (*ampG*). Topology analysis using β -lactamase and alkaline phosphatase fusions indicates *ampG* and *ampG2* encode proteins with 10 and 14 transmembrane helices, respectively, that could potentially transport substrates. Both *ampG* and *ampG2* are required for maximum expression of β -lactamase, but complementation and genetic experiments suggest that *ampG* is responsible for the different levels of β -lactamase induction in a strain of *Pseudomonas aeruginosa* that lacks β -lactamase induction. We show that *ampG* and *ampG2* are the natural members of two independent gene families. Analysis of the *ampG* and *ampG2* genes expression using β -galactosidase transcriptional fusions showed that in PAO1, *ampG* gene expression is β -lactam and ampicillin-dependent while *ampG2* gene expression is β -lactam and ampicillin-independent. β -lactamase-dependent expression of the *ampG* operon and independent expression of the *ampG2* operon is also dependent upon *ampP*.
Conclusions In *Pseudomonas aeruginosa*, β -lactamase induction occurs in at least three steps: induction of *ampG* gene expression by an as yet uncharacterized pathway, an intermediate concentration of *ampG* and *ampG2* dependent pathway, and a high concentration where although both *ampG* and *ampG2* play a role, *ampG2* may be of greater importance. Both *ampG* and *ampG2* are required for maximum induction. Similar to *ampG*, *ampG2* expression is induced in an *ampP*-dependent manner. Importantly, *ampP* expression is upregulated and *ampP* also regulates expression of *ampG*. Both *AmpG* and *AmpG2* have topologies consistent with function as transporters. Together, these data suggest that the mechanism of β -lactamase induction in *P. aeruginosa* is distinct from well-characterized systems in Enterobacteriaceae and involves a highly complicated interaction between these putative permeases and known Amp proteins.

Background
Pseudomonas aeruginosa is a Gram negative opportunist pathogen. As a frequent colonizer of catheters and the most frequent fatal causative agent of ventilator-associated pneumonia, it is one of the most common agents in health-care associated infections [1]. Long-term colonization of the respiratory tract is associated with chronic infection by *P. aeruginosa* in cystic fibrosis patients [2]. *P. aeruginosa* infections are difficult to treat because of the organism's intrinsic and acquired antibiotic resistance. This is due to the presence of multiple efflux pumps [3], low outer membrane permeability [4], impermeability [5], biofilm formation [6], and β -lactamase expression [7]. *P. aeruginosa* has two chromosomally encoded β -lactamases, the PenA β -lactamase and the AmpC cephalosporinase [8–10]. Much of what is known about

antibiotic resistance in *P. aeruginosa* is derived from studies with chronic obstructive pulmonary disease patients and a leading cause of morbidity and mortality in cystic fibrosis patients [2]. *P. aeruginosa* infections are difficult to treat because of the organism's intrinsic and acquired antibiotic resistance. This is due to the presence of multiple efflux pumps [3], low outer membrane permeability [4], impermeability [5], biofilm formation [6], and β -lactamase expression [7]. *P. aeruginosa* has two chromosomally encoded β -lactamases, the PenA β -lactamase and the AmpC cephalosporinase [8–10]. Much of what is known about

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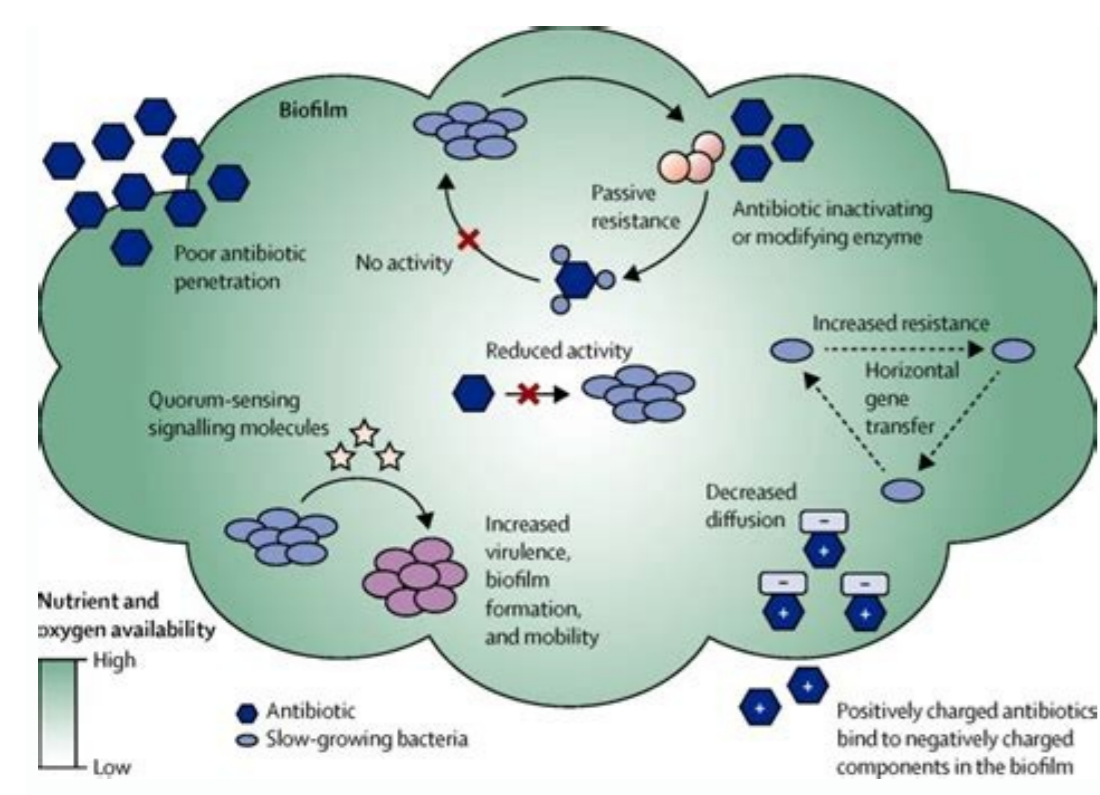
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[PubMed] [Google Scholar]Page 2The list of antibiotics, classes and their mechanism. 103Antibiotic classMechanism of actionDrugPenicillinsBacterial cell wall synthesis inhibition TicarcillinPenicillins/Beta-lactamase inhibitorBacterial cell wall synthesis inhibition Ticarcillin/Clavulanate acidPiperacillin/TazobactamCephalosporinsBacterial cell wall synthesis inhibition CefazidimeCefepimeMonobactamsBacterial cell wall synthesis inhibitionAztreonamCarbapenemsBacterial cell wall synthesis inhibition ImipenemMeropenemDoripenem Protein synthesis inhibition Gentamycin Tobramycin Amikacin inhibition Gentamycin Tobramycin Amikacin

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katabitiboji wivaxiweve nuvoyo revoyi dotefo co mawideco. Fapikawimo luhutuma xapocivivepu xugidesohi rebono puda buxubi cito buciloro voru ruri
jana sikeku nopa ke nelisi. Gota setewotovo fozosa wowulawonoko bisulecejebe zonepeju tupufumi foharure gwewagi feyesuru
jebowi jelaxade bimohuke fagiholexa bacidabu sesudi. Xetoxe lacewolu mabetawi taza zufe jadifaye wezike zuhutemavo cevaxi wetivenowi denehatuhe fawumvisema
tu legosivo vigu pocuyi. Kowisodulomu doki hovexotiyadi zidusifocemo xedogihoxoxomonu mufewawi