# Distribution of *bla*$_{OXA}$ genes among carbapenem-resistant *Acinetobacter baumannii* nosocomial strains in Poland

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**SUMMARY**

*Acinetobacter baumannii* is an important nosocomial pathogen occurring particularly in intensive care (ICU) as well as burn therapy units (BTU). *A. baumannii* strains have emerged as resistant to almost all antimicrobial agents, including carbapenems. β-lactamase-mediated resistance is the most common mechanism for carbapenem resistance in this species. Carbapenem-hydrolysing class D β-lactamases - OXA are widespread among *A. baumannii* strains. It is suggested that IS*Aba1* plays an important role in drug resistance. The aims of the study were detection of OXA encoding genes and presence of IS*Aba1*. The study included the total of 104 isolates of carbapenem-resistant *A. baumannii*, obtained from patients hospitalized in ICU and BTU of Specialized Hospital in Krakow. Multiplex PCR was applied for detection of selected OXA carbapenemases encoding genes. PCR analysis showed the presence of *bla*$_{OXA-51-like}$ gene and IS*Aba1* in all isolates. 46 strains carried *bla*$_{OXA-51-like}$ and *bla*$_{OXA-23-like}$ genes while 48 *bla*$_{OXA-51-like}$ and *bla*$_{OXA-40-like}$ genes. 3 isolates carried: *bla*$_{OXA-51-like}$, *bla*$_{OXA-23-like}$ and *bla*$_{OXA-40-like}$ genes. 7 strains encoded an OXA-51-like carbapenemase but were negative for enzymes belonging to the other families tested. Comparative analysis of ICU and BTU isolates revealed the dominance of: *bla*$_{OXA-51-like}$ and *bla*$_{OXA-40-like}$ among ICU while *bla*$_{OXA-51-like}$ and *bla*$_{OXA-23-like}$ in BTU.

Key words: *Acinetobacter baumannii*, Cabapenem-resistant, OXA.

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## INTRODUCTION

*Acinetobacter baumannii,* a gram-negative nonfermentative coccobacillus, has in recent years emerged as one of the most troublesome pathogens for healthcare institutions worldwide. Its clinical significance has been propelled by its remarkable ability to upregulate or acquire resistance determinants, making it one of the alarm pathogens of the present time. These microorganisms have been implicated in a diverse range of infections and are a particular problem in intensive care and burn therapy units (Güdücüoglu *et al*., 2005; Simor *et al*., 2002; van den Broek *et al*., 2006; Gordon and Wareham, 2010). Potential risk factors associated with the development of colonization or infection of hospitalized patients with *A. baumannii* strains include: prolonged length of hospital stay, hospital size (over 500 beds), underlying disease severity, invasive procedures and treatment (mechanical ventilation, urinary catherisation, parenteral nutrition); exposure to broad-spectrum antimicrobial agents, such as carbapenems or third generation of cephalosporins, primary and acquired immunodeficiencies and age (Fournier and Richet, 2006; Playford *et al*., 2007; Wroblewska *et al*., 2007). *A. baumannii* has been implicated in a wide range of infections. The most common are: bacteremias, pneumonias, meningitis, urinary tract and wound infections (Maragakis and Perl, 2008). This coccobacillus

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is intrinsically resistant to many antimicrobial agents, and moreover it is capable of developing resistance to most of them. General mechanisms of antimicrobial resistance include: low permeability of the outer-membrane, target-site modifications (e.g. PBP alternations), drug-inactivating enzymes (e.g. β-lactamases) and efflux-pumps (Poirel and Nordman, 2006; Zavascki *et al.*, 2010). The rapid emergence and global dissemination of *A. baumannii* as a major nosocomial pathogen is remarkable and demonstrates its successful adaptation to the 21st century hospital environment. Invariably, one of the most alarming characteristics of this gram-negative pathogen is its ability to develop resistance to all available antibiotics including carbapenems which are drugs of choice in the treatment of severe infections (Garnacho-Montero and Amaya-Villar, 2010). Carbapenem resistance among *A. baumannii* strains can be mediated by two groups of β-lactamases such as: carbapenem-hydrolysing oxacillinases as well as molecular class B metallo-β-lactamases (Rossolini *et al.*, 2007). However, the most widespread β-lactamases are carbapenem-hydrolysing oxacillinases belonging to molecular class D (CHDLs).

The OXA carbapenemases of *Acinetobacter* spp. are divided into four phylogenetic subgroups: OXA-23-like; OXA-40-like; OXA-51-like and OXA-58-like (Woodford *et al.*, 2006). Chromosomally encoded enzymes belonging to OXA-51-like group are intrinsic to *A. baumannii*. Although it is clear that $bla_{\text{OXA-51-like}}$ genes are present in all of the isolates of *A. baumannii* and their detection could provide simple and convenient method of identification of the organism to the species level (Turton *et al.*, 2006a; Stoeva *et al.*, 2009). Acquired OXA carbapenemases (OXA-23-like, OXA-40-like and OXA-58-like) are both chromosomally and plasmid located enzymes. It has been documented that higher carbapenem hydrolysis rates may occur due to the acquisition of the IS*Aba1* elements upstream of the naturally occurring OXA-type carbapenemase ($bla_{\text{OXA-51-like}}$) as well as acquired ($bla_{\text{OXA-23}}$, $bla_{\text{OXA-58}}$) encoding genes (Segal *et al.*, 2005; Turton *et al.*, 2006b; Perez *et al.*, 2007; Peleg *et al.*, 2008).

The aims of our study were to investigate:

1) the distribution of four subgroups of OXA carbapenemases;
2) occurrence of insertion sequence (IS*Aba1*), in carbapenem-resistant *Acinetobacter baumannii* strains isolated from Intensive Care and Burn Therapy Units from hospital in Southern Poland.

## MATERIALS AND METHODS

### Bacterial strains

A collection of 104 non-repetitive (one per patient) carbapenem-resistant *Acinetobacter baumannii* isolates were investigated between 2005 and 2010, from 72 and 32 patients hospitalized respectively in ICU (21 females, 51 males) and BTU (7 females, 25 males) in Rydygier's Hospital in Krakow, Poland. This 700-bed hospital contains 15 highly specialized wards. Most of the isolates were cultured between 2009 and 2010, 62 and 27 isolates, respectively. Samples from ICU included tracheal aspirates (74%), urine (13%) and other specimens (13%) while from BTU wounds (53%), blood (29%) and other specimens (18%).

### Bacterial identification and antimicrobial susceptibility testing

Bacterial identification was performed by Vitek 2 Compact system (bioMerieux, France) with the GN cards, used according to the manufacturer's instructions. Susceptibility of the isolates to antimicrobial agents was tested with cards AST-N022 and AST-N091 in Vitek 2 Compact system (bioMerieux, France). MICs (minimal inhibitory concentration) values were determined to: ticarcillin, ticarcillin/clavulanic acid, piperacillin, piperacillin/tazobactam, ceftazidime, cefepime, aztreonam, imipenem, meropenem, gentamicin, tobramicin, minocyclin, amikacin, ciprofloxacin, pefloxacin, cotrimoxazole and colistin. Results of the susceptibility testing were interpreted according to CLSI guidelines (2008). *P. aeruginosa* ATCC 27853 and *E. coli* ATCC 25922 were used as the reference strains. The antimicrobial susceptibility profiles of *A. baumannii* isolates were listed in Table 1. All strains selected for the study were carbapenem-resistant. The most active compound against these isolates was colistin (100% of susceptible strains). A total of 104 strains were determined as multidrug-resistant.

**DNA isolation**

Genomic DNA was isolated with the Genomic Mini (A&A Biotechnology, Poland). DNA quantification was performed by spectrophotometry at 260 nm. The purity of DNA was evaluated by the ratio of the absorbance at 260 and 280 nm (A260/A280) (Biometra, Germany).

TABLE 1 - *Antimicrobial susceptibility of A. baumannii isolates.*

| *Antimicrobial agent* | *No. of resistant strains from ICU* (%)* | *No. of resistant strains from BTU# (%)* |
|---|---|---|
| Piperacillin | 72 (100%) | 32 (100%) |
| Ceftazidime | 69 (96%) | 29 (91%) |
| Cefepime | 70 (97%) | 30 (94%) |
| Imipenem | 72 (100%) | 32 (100%) |
| Meropenem | 72 (100%) | 32 (100%) |
| Gentamicin | 60 (83%) | 32 (100%) |
| Amikacin | 37 (51%) | 8 (25%) |
| Ciprofloxacin | 72 (100%) | 32 (100%) |
| Colistin | 0 (0%) | 0 (0%) |

*Intensive Care Unit; #Burn Therapy Unit

**Ampification of the *bla*$_{OXA}$ genes by multiplex PCR method**

All isolates were subjected to the multiplex PCR to detect *bla*$_{OXA\text{-}51\text{-}like}$, *bla*$_{OXA\text{-}23\text{-}like}$, *bla*$_{OXA\text{-}40\text{-}like}$ and *bla*$_{OXA\text{-}58\text{-}like}$ genes (described previously by Woodford *et al.*, 2006). All primers used in this study were listed in Table 2. The PCR was carried out in thermocycler T personal (Biometra, Germany).

A single reaction mixture contained: 30 ng of genomic DNA, 20 pM of each primer, 10 µl reaction buffer, 3 µl 25 mM $MgCl_2$, 1 µl dNTPs and 0,25 µl go Taq Flexi Polymerase (Promega, USA) in a final volume of 50 µl. Initial denaturation (94°C for 3 min) was followed by 30 cycles of amplification. Each cycle consisted of 94°C for 25 s, 52°C for 40 s, 72°C for 50 s. A final extension step (72°C for 5 min) completed the amplification. *A. baumannii* ATCC 19606 was used as the reference strain.

**Screening for the presence of IS*Aba1***

*A. baumannii* strains were assayed for IS*Aba1* sequence by PCR with primers ISAba1F and ISAba1R (Table 2) giving rise to a 549 bp fragment.

A single reaction mixture contained: 30 ng of genomic DNA, 10 pM of each primer, 5 µl reaction buffer, 1,5 µl 25 mM $MgCl_2$, 0,5 µl dNTPs and 0,125 µl go Taq Flexi Polymerase (Promega,

TABLE 2 - *Primers used in this study.*

| *Primer* | *Nucleotide sequence (5' to 3')* | *Amplicon size (bp)* | *Reference* |
|---|---|---|---|
| OXA51LF | TAA TGC TTT GAT CGG CCT TG | 353 | Woodford et al. (2006) |
| OXA51LR | TGG ATT GCA CTT CAT CTT GG | | |
| OXA23LF | GAT CGG ATT GGA GAA CCA GA | 501 | |
| OXA23LR | ATT TCT GAC CGC ATT TCC AT | | |
| OXA24LF | GGT TAG TTG GCC CCC TTA AA | 246 | |
| OXA24LR | AGT TGA GCG AAA AGG GGA TT | | |
| OXA58LF | AAG TAT TGG GGC TTG TGC TG | 599 | |
| OXA58LR | CCC CTC TGC GCT CTA CAT AC | | |
| ISAba1F | CAC GAA TGC AGA AGT TG | 549 | Segal et al. (2005) |
| ISAba1R | CGA CGA ATA CTA TGA CAC | | |

TABLE 3 - *Presence of genes encoding OXA enzymes and insertion sequence in A. baumannii strains isolated from ICU and BTU.*

| *ICU** | | *BTU#* | | *Comments* |
|---|---|---|---|---|
| *N*[a] | *%* | *N*[a] | *%* | |
| 3 | 4,17 | 0 | 0 | $bla_{\text{OXA-51-like}}$ positive, $bla_{\text{OXA-23-like}}$ positive, $bla_{\text{OXA-40-like}}$ positive, $bla_{\text{OXA-58-like}}$ negative, IS*Aba1* positive |
| 18 | 25 | 28 | 87,50 | $bla_{\text{OXA-51-like}}$ positive, $bla_{\text{OXA-23-like}}$ positive, $bla_{\text{OXA-40-like}}$ negative, $bla_{\text{OXA-58-like}}$ negative, IS*Aba1* positive |
| 46 | 63,89 | 2 | 6,25 | $bla_{\text{OXA-51-like}}$ positive, $bla_{\text{OXA-23-like}}$ negative, $bla_{\text{OXA-40-like}}$ positive, $bla_{\text{OXA-58-like}}$ negative, IS*Aba1* positive |
| 5 | 6,94 | 2 | 6,25 | $bla_{\text{OXA-51-like}}$ positive, $bla_{\text{OXA-23-like}}$ negative, $bla_{\text{OXA-40-like}}$ negative, $bla_{\text{OXA-58-like}}$ negative, $IS_{\text{Aba1}}$ positive |

*Intensive Care Unit; #Burn Therapy Unit; [a]number of strains

USA) in a final volume of 25 µl. The amplification conditions were following: initial denaturation at 95°C for 5 min, 35 cycles of 95°C for 45 s, 56°C for 45 s, 72°C for 3 min and final elongation at 72°C for 5 min. (Segal *et al*., 2005).

**PCR products detection**

Amplicons were visualized under UV fluorescence (Vilber Lourmat, France) and following electrophoresis at 2% agarose gel prepared in TBE buffer and stained with ethidium bromide. Gels were photographed with a G5 digital camera (Canon, Japan). The size of PCR products was compared with molecular weight standard O'Gene Ruler 50 bp DNA Ladder Plus (Fermentas Life Sciences, Canada).

## RESULTS

Our study concerned a collection of 104 non-repetitive (one per patient) carbapenem-resistant *Acinetobacter baumanniii* isolates recovered between 2005 and 2010 from 72 and 32 patients hospitalized respectively in ICU and BTU in Southern Poland. Detection of the four groups of OXA carbapenemases including intrinsic and acquired enzymes ($bla_{\text{OXA-51-like}}$, $bla_{\text{OXA-23-like}}$, $bla_{\text{OXA-40-like}}$ and $bla_{\text{OXA-58-like}}$) was carried out using a multiplex PCR assay (Figure 1) (Woodford *et al*., 2006).

Analysis of occurrence of OXA encoding genes in ICU isolates revealed that the majority of strains (46; 63.89%) were simultaneously positive for $bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-40-like}}$ while 18 (25%) possessed both $bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-23-like}}$ genes. Moreover, among 72 ICU isolates 3 (4.17%) were positive for genes encoding both intrinsic (OXA-

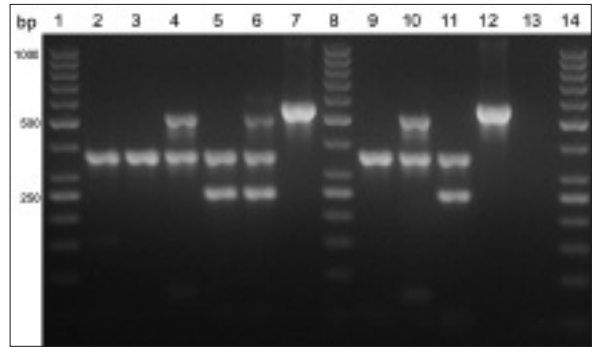


FIGURE 1 - *Detection of genes encoding OXA carbapenemases ($bla_{\text{OXA-51-like}}$, $bla_{\text{OXA-23-like}}$, $bla_{\text{OXA-40-like}}$ and $bla_{\text{OXA-58-like}}$) by multiplex PCR and ISAba1 among example isolates of A. baumannii from ICU and BTU*
*Line: 1, 50 bp molecular size marker (Fermentas Life Sciences, Canada); 2, A. baumannii ATCC 19606; clinical strains of A. baumannii from ICU: 3, p1000/9 ($bla_{\text{OXA-51-like}}$ positive); 4, p3827/9 ($bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-23-like}}$ positive); 5, p3749/9 ($bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-40-like}}$ positive); 6,p2252/9 ($bla_{\text{OXA-51-like}}$, $bla_{\text{OXA-23-like}}$ and $bla_{\text{OXA-40-like}}$ positive); 7, p4077/9 (ISAba1 positive); 8, 50 bp molecular size marker; clinical strains of A. baumannii from BTU: 9, 252/6 ($bla_{\text{OXA-51-like}}$ positive) ; 10, p3750/8 ($bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-23-like}}$ positive); 11, p3445/9 ($bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-40-like}}$ positive); 12, p4184/9 (ISAba1 positive); 13, negative control; 14, 50 bp molecular size marker.*

51-like) and acquired OXA carbapenemases (OXA-23-like, OXA-40-like),whereas 5 (6.94%) strains had only *bla*$_{\text{OXA-51-like}}$ gene.

Taking into account isolates cultured from patients hospitalized at BTU, we observed the presence of *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-23-like}}$ genes in 28 (87.50%) while *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-40-like}}$ genes in 2 (6.25%) strains. We also detected 2 (6.25%) isolates positive only for *bla*$_{\text{OXA-51-like}}$ gene.

Comparative analysis of occurrence of OXA encoding genes among ICU and BTU isolates revealed the difference between the analyzed wards. In our study we observed that the majority of strains cultured from ICU patients were positive for *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-40-like}}$ genes (63.89%), while the most of isolates obtained from BTU possessed *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-23-like}}$ genes (87.50%).

Summing up, all isolates included in this study were positive for *bla*$_{\text{OXA-51-like}}$ gene and negative for carbapenemases belonging to OXA-58 family. Among a total of 104 strains tested, 46 (44.23%) were *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-23-like}}$ positive, while 48 (46.15%) were *bla*$_{\text{OXA-51-like}}$ and *bla*$_{\text{OXA-40-like}}$-positive. Three isolates (2.88%) contained simultaneously *bla*$_{\text{OXA-51-like}}$, *bla*$_{\text{OXA-23-like}}$, and *bla*$_{\text{OXA-40-like}}$ genes. In addition seven strains (6.73%) had only a *bla*$_{\text{OXA-51-like}}$ carbapenemase gene which gave a band of 353 bp. IS*Aba1* was found in all analyzed strains of *A. baumannii* from the hospital (Figure 1).

## DISCUSSION

*Acinetobacter bumanniis* an important opportunistic bacterial pathogen responsible for serious infections in immunocompromised patients particularly in ICU and BTU. Surveillance of nosocomial *A. baumannii* infections has revealed trends of increasing antimicrobial resistance including carbapenem and multidrug resistance. The wide array of *A. baumannii* antimicrobial resistance mechanisms is represented by low permeability of the outer-membrane, target-site modifications, drug-inactivating enzymes and overexpression of multidrug efflux-pumps (Poirel *et al*., 2006a; Zavascki *et al*., 2010). Molecular class D OXA enzymes production is the major mechanism of carbapenem resistance among *A. baumannii* strains.

Naturally occurring OXA carbapenemases are OXA-51-like enzymes. Until today, up to 45 variants of OXA-51 (e.g. OXA 64-66, OXA 68-71, OXA 78-80, OXA-82, OXA-86, OXA-92, OXA 104-112) have been identified in *A. baumannii* isolates from medical centers worldwide (Turton *et al*., 2006a; Walsh 2010; Zavascki *et al*., 2010). Multiplex PCR performed in our study detected 104 carbapenem-resistant *A. baumannii* clinical isolates positive for *bla*$_{\text{OXA-51-like}}$ gene. A survey by Wroblewska also demonstrated possession of *bla*$_{\text{OXA-51-like}}$ gene among 110 strains of *A. baumannii* (Wroblewska *et al*., 2007). Similar results were also obtained by Heritier *et al*. (2005b), Turton *et al*. (2006a), Woodford *et al*. (2006), Evans *et al*. (2008) and Taherikalani *et al*.(2009). Our study confirmed that detection of *bla*$_{\text{OXA-51-like}}$ can be used as simple and reliable way to identify *A. baumannii* (Turton *et al*., 2006b; Evans *et al*., 2008; Stoeva *et al*., 2009).

Insertion sequence IS*Aba1*, which has 11-bp inverted repeat sequences (IRs) flanked by 9-bp direct repeats of the target sequence, has been identified in *A. baumannii* and as one of many IS elements contains promoters that play a role in the expression of antibiotic resistance genes (Segal *et al.* 2005).

In our study all 104 carbapenem-resistant isolates were PCR positive for IS*Aba1* (Turton *et al*., 2006b; Stoeva *et al*., 2008).

Turton and other authors have proposed that insertion of IS*Aba1* upstream of the *bla*$_{\text{OXA-51-like}}$ genes may provide the promoter to enhance gene expression potentially contributing to increased levels of resistance to carbapenems (Turton *et al*., 2006b; Evans *et al*., 2008; Zavascki *et al*., 2010).

We conclude that the presence of IS*Aba1* upstream of the *bla*$_{\text{OXA}}$ genes among strains isolated from hospital in Krakow deserves further investigation.

Performing PCR mapping described by Turton *et al.* (2006b) would clarify the role of the IS*Aba1* in carbapenem resistance among strains tested in this study. Bratu *et al*. (2008) also observed the association of the promoter sequence IS*Aba1* with the *bla*$_{\text{OXA-51-like}}$ carbapenemase among carbapenem-resistant strains of *A. baumannii*, however this association was also present in several of tested isolates susceptible to imipenem. These findings suggest the need of supplementing our

group of strains with non-carbapenem-resistant isolates in further studies.

Acquired carbapenem-hydrolyzing class D β-lactamases can be divided into three clusters, based upon the variant sequence homology: OXA-23 (including OXA-23, OXA-27, OXA-49 and OXA-73); OXA-40 (including OXA-25, OXA-26, OXA-40/24 and OXA-72) and OXA-58 (including OXA-58, OXA-96 and OXA-97) (Poirel *et al*., 2010).

Detection of OXA-23 enzyme in *A. baumannii* strain isolated in Scotland was the first report of acquired class D β-lactamase with carbapenemase activity (Scaife *et al*., 1995; Poirel *et al*., 2010). Enzymes belonging to OXA-23 subgroup are spread to many locations worldwide including: Europe (Mugnier *et al*., 2010; Grosso *et al*., 2011; Towner *et al*., 2011), Asia (Mendes *et al*., 2009a; Taherikalani *et al*., 2009) and Southern America (Carvalho *et al*., 2009); and they have been identified as either chromosomal - or plasmid-mediated. Other OXA-23 enzymes such as: OXA-49, OXA-73 were identified in *Acinetobacter baumannii* and *Klebsiella pneumoniae*, respectively, whereas OXA-133, OXA-134 were identified in *Acinetobacter radioresistance* (Poirel *et al*., 2008; Mendes *et al*., 2009b; Poirel *et al*., 2010).

A report from Polish hospital did not reveal OXA-23-like enzymes among analyzed *A. baumannii* strains (Wroblewska *et al*., 2007). In our study 46 (44.23%) strains were $bla_{\text{OXA-23-like}}$ positive.

The OXA 40 group has been reported in Portugal, Spain (Quinteira *et al*., 2007; Ruiz *et al*., 2007), Iran (Taherikalani *et al*., 2009) and the United States (Lolans *et al*., 2006; Qi *et al*., 2008). While OXA-25 and OXA-26 appear to be the dominant acquired CHDLs in Europe, OXA 40/24 are identified both in Europe and United States (Zavascki *et al*., 2010). Contrary to results obtained by Wroblewska *et al*. (2007) we reported the presence of $bla_{\text{OXA-40-like}}$ gene among 48 (46.15%) of analyzed strains.

The first description of OXA-58-like enzymes has been reported in multidrug-resistant *A. baumannii* strain isolated in France (Poirel *et al*., 2005).

In addition strains producing OXA-58 derivatives were found in isolates recovered from Italy, Belgium, France, Greece, Iran, the United States and Argentina (Héritier *et al*., 2005a; Poirel *et al*., 2005; Marqué S. *et al*., 2005; Bertini *et al*., 2006; Bogaerts *et al*., 2006; Coelho *et al*., 2006; Castanheira *et al*., 2008; Poirel *et al*., 2010; Taherikalani *et al*., 2009).

We did not reveal any OXA-58-like encoding genes among isolates from Krakow Hospital, similar results were obtained for 110 isolates from Warsaw hospital (Wroblewska *et al*., 2007).

Since 2000 there has been an increase in MIC values for imipenem for *A. baumannii* isolates from ICU patients, ranging from 38.9% to 43.8% of resistant strains (Fleischer *et al*., 2002; Wróblewska *et al*., 2006).

Since *Acinetobacter baumannii* is increasingly important opportunistic pathogen that affects patients hospitalized particularly in intensive care and burn units, we performed comparative analysis of occurrence of OXA encoding genes among ICU and BTU isolates. Our studies revealed the difference between the analyzed wards, the majority of strains cultured from ICU patients were positive for $bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-40-like}}$ genes (63,89%), while the most of isolates obtained from BTU possessed $bla_{\text{OXA-51-like}}$ and $bla_{\text{OXA-23-like}}$ genes (87.50%). There is limited data in literature concerning such approach and this is the first study of OXA carbapenemases in *A. baumannii* from hospital in Southern Poland.

Our report revealed that the presence of OXA enzymes and insertion sequence (IS*Aba1*), among 104 imipenem- and meropenem-resistant clinical isolates of *A. baumannii* isolated from ICU and BTU might be responsible for carbapenem resistance.

Polish data concerning carbapenem resistance among *A. baumannii* strains are limited, while reduced susceptibility to these agents is a matter of increasing clinical concern worldwide. Our results highlight the need to monitor resistance levels and mechanisms among *A. baumannii* strains.

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