

Comparison of BDPhoenix and VITEK2 automated antimicrobial susceptibility test systems for extended-spectrum beta-lactamase detection in *Escherichia coli* and *Klebsiella* species clinical isolates

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Received 16 April 2002; accepted 21 August 2002

Abstract

The present study compares the ability to detect extended-spectrum β -lactamases (ESBL) among a collection of 34 ESBL producing clinical isolates belonging to *Escherichia coli* and *Klebsiella* species with two new rapid susceptibility and identification instruments—VITEK2 (bioMérieux, Marcy l'Etoile, France) vs. BDPhoenix (BD Biosciences, Sparks, MD). ESBL content in these isolates was previously characterized on the basis of PCR amplification and sequencing results which were used as the reference method in our evaluation. BDPhoenix correctly determined the ESBL outcome for all strains tested (100% detection rate), whereas VITEK2 was not able to detect the ESBL status in 5 isolates (85% detection rate). Detailed analysis revealed that the discrepancies were mainly observed with 'difficult-to-detect' strains. Misidentification was either due to low oximino cephalosporin MIC in these strains or was associated with pronounced 'cefotaximase' or 'ceftazidimase' phenotypes. *Klebsiella oxytoca* chromosomal β -lactamase (K1) is phenotypically quite similar to ESBL enzymes. In order to evaluate whether the K1 and ESBL enzymes could be discriminated, we expanded our analysis by 8 clinical *K. oxytoca* strains with K1 phenotypes. VITEK2 gave excellent identification of these strains whereas 7 out of 8 were falsely labeled ESBL-positive by the BDPhoenix system. © 2003 Elsevier Science Inc. All rights reserved.

1. Introduction

Extended-spectrum β -lactamases (ESBLs) constitute an ever-growing class of mainly plasmid-mediated β -lactamases found in Gram-negative bacilli. They confer resistance to broad spectrum cephalosporins (e.g., ceftazidime, cefotaxime, ceftriaxone and cefpodoxime) and/or the monobactam aztreonam, but do not affect cephamycins, carbapenems and β -lactamase inhibitors such as clavulanic acid (Livermore, 1995; Bradford, 2001). Although most ESBLs confer resistance to one or more of the oximino cephalosporins, some enzymes do not always elevate the MICs to levels high enough to be called resistant according to the breakpoints advocated by the National Committee for Clinical Laboratory Standards (NCCLS, 2000; NCCLS, 2002), despite clear demonstration of clinical failures of the drug if used in therapy (Paterson et al., 2001; Kim et al., 2002). Thus, the accurate and reliable detection of the ESBL mech-

anism is a prerequisite for successful therapeutic management.

For reasons of rapidity and convenience, integrated systems which automatically perform rapid identification and antimicrobial susceptibility testing are increasingly used in daily routine (Ferraro & Jorgensen, 1999). For most current instruments an ESBL prediction has been integrated into the automated procedure. Two detection strategies are in competition: (i) ESBL prediction from the MIC pattern (Sanders et al., 2000) or (ii) using ceftazidime or cefpodoxime as an indicator drug, followed by screening for synergy between oximino cephalosporins and clavulanic acid as recommended by the NCCLS (NCCLS, 2002). However, in order to promote more rapid results, the growth media and conditions as well as the testing procedures in cards and panels of the automated systems are often modified compared with conventional methods (Ferraro & Jorgensen, 1999). Up to now, valid data on the ability of such modifications to accurately detect the ESBL mechanism is still sparse. A second question that needs to be answered in this context concerns the discriminatory power of the automated procedures; currently, there is too little information available on

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how well these cards and panels can separate between ESBL and other resistance mechanisms that mimic the ESBL phenotype (e.g., K1 enzyme in *Klebsiella oxytoca*).

The present study was conducted to compare the performance of two new automated instruments, VITEK2 (bioMérieux, Marcy l'Etoile, France) and BDPhoenix (BD Biosciences, Sparks, MD), in detecting the ESBL status in a challenge set of genetically characterized ESBL carriers (22 *Klebsiella pneumoniae*, 9 *Escherichia coli* and 3 *K. oxytoca*) and to assess discrimination of the automated microbiology systems between true ESBL content and an ESBL-simulating phenotype (8 *K. oxytoca* isolates with K1 β -lactamase production).

2. Materials and methods

2.1. Bacterial isolates, culture conditions, and inoculum preparation

A total of 42 clinical isolates belonging to *E. coli* and *Klebsiella* species, 34 with ESBL and 8 with K1 enzymes, were included in the present study. Isolates were subcultured once on Columbia sheep blood agar before being tested. The inoculum preparation was performed according to the manufacturer's suggested procedures, using a densitometer device for standardization (McFarland 0.5–0.6) and paying careful attention to use fresh colonies. The inoculated cards and panels were placed into their respective workstation for incubation and reading and were interpreted by the incorporated computerized expert system (VITEK2: Advanced Expert System, AES; BDPhoenix: BDXpert System). Species identification was performed by appropriate routine laboratory methods, such as the ID32E galleries (bioMérieux, Marcy l'Etoile, France), BBL Enterotube II and BBL Crystal E/NF ID System (Becton Dickinson Microbiology Systems, Cockeysville, MD). MICs were used as reference values when evaluating disagreements observed with the automated systems. MICs were determined by standard broth microdilution tests using commercial trays MICRONAUT-S (Merlin Diagnostika, Bornheim, Germany) and Mueller-Hinton broth (Difco Laboratories, Detroit, MI) according to the guidelines of the NCCLS (NCCLS, 2000; NCCLS, 2002). Quality control was performed by running *E. coli* ATCC 25922, *K. pneumoniae* DSM 30104, *K. pneumoniae* ATCC 700603 (SHV-18), and *K. oxytoca* DSM 5175 for each new lot of cards, panels or MIC trays.

2.2. Characterization of ESBL carriers

Clinical ESBL isolates belonging to *E. coli* and *Klebsiella* spp. were collected between 1998 and 2001 from individual patients at the Universitätsklinikum Hamburg-Eppendorf (Hamburg, Germany). Only one strain of a given species and susceptibility pattern per patient was accepted.

All strains fulfilled the screening recommendations of the NCCLS document M100-S12 (NCCLS, 2002), i.e., they produced inhibition zone diameters of ≤ 27 mm for cefotaxime (30 μ g), ≤ 27 mm for aztreonam (30 μ g), ≤ 22 mm for ceftazidime (30 μ g), or ≤ 25 mm for ceftriaxone (30 μ g). The ESBL status was confirmed by a positive synergy test using the technique recommended by document M100-S12 (NCCLS, 2000; NCCLS, 2002); i.e., in these strains the zone diameters for either ceftazidime plus clavulanic acid or cefotaxime plus clavulanic acid were at least 5 mm larger than the zone diameters for ceftazidime or cefotaxime alone.

2.3. PCR amplification and DNA sequencing

ESBL producing isolates were grouped on the basis of PCR amplification and sequencing results. Template DNA was prepared by suspending four colonies from overnight cultures in 0.2 mL of H₂O, and heating to 100°C for 10 min. PCRs were performed in 50 μ L with the DyNAzyme DNA Polymerase Kit (Finnzymes, Espoo, Finland), using 1.5 mM MgCl₂, 15 pmol of each primer and 1 unit DyNAzyme DNA polymerase together with 5 μ L of template DNA. Primers for amplification of the TEM gene were as follows: OT-3, ATG AGT ATT CAA CAT TTC CG (corresponding to nucleotides 3950–3967 in the sequence published under accession number J01749 (Sutcliffe, 1978), and OT-4, TTA CCA ATG CTT AAT CAG TGA GG (nucleotides 4810–4788 in the same sequence) giving an amplification product of 860 bp; those for the SHV gene were: OS-5, TTA TCT CCC TGT TAG CCA CC (nucleotides 28–47 in the sequence listed under accession number AF148850 (Bradford, 1999) and OS-6, GAT TTG CTG ATT TCG CTC GG (nucleotides 824–805 in the same sequence) giving a product of 796 bp; those for the CTX-M gene were: CTX-M10for, GCA GCA CCA GTA AAG TGA TGG (nucleotides 277–297 in the sequence published under accession number X92506 (Bauernfeind et al., 1996), and CTX-M10rev, GCG ATA TCG TTG GTG GTA CC (nucleotides 811–192 in the same sequence) giving a product of 534 bp. Amplification parameters included a 4-min denaturation at 94°C, followed by 35 cycles of denaturation (94°C for 30 sec), annealing (60°C for 30 sec), and extension (72°C for 1 min), ending in a final extension period of 72°C for 20 min. PCR-amplified DNA fragments were directly sequenced on both strands by using an automated DNA-sequencing procedure (ABI Prism 310 DNA Sequencer, Applied Biosystems, Foster City, CA) involving fluorescent dye-labeled terminators (Applied Biosystems, Foster City, CA). Sequencing primers were the same as for PCR amplification.

2.4. Characterization of *K. oxytoca* K1 producers

Eight routine *K. oxytoca* strains met the screening criteria for ESBL production according to NCCLS document M100-S12 (NCCLS, 2002) but were negative in both cla-

vulanate synergy test and PCR amplification of ESBL genes. These isolates had reduced susceptibility, or were resistant, to cefuroxime, ceftriaxone, and aztreonam, whereas ceftazidime and cefotaxime consistently retained susceptibility against these strains. Since this is the typical phenotype for K1 enzyme (Livermore, 1995), these strains were attributed to this mechanism as the primary cause of the resistance observed.

2.5. Antimicrobial susceptibility testing cards and panels

The card and panels used for this study were standard European configurations for members of the family *Enterobacteriaceae* and shared a comparable composition of β -lactam antimicrobials and concentration ranges (in $\mu\text{g}/\text{ml}$) as follows: (i) AST-N011 for VITEK2 (ampicillin, 4-32; ampicillin-sulbactam, 4/2-32/16; cefazolin, 4-64; cefepime, 1-16; cefixime, 0.25-2; cefotaxime, 1-32; ceftazidime, 1-32; cefuroxime, 2-32; imipenem, 2-16; piperacillin, 4-64; piperacillin-tazobactam, 4/4-128/4) and (ii) NMIC/ID-6 Combo Panel for BDPhoenix (ampicillin, 4-16; amoxicillin-clavulanate, 4/2-16/8; aztreonam, 2-16; cefazolin, 4-16; cefotaxime, 2-32; cefpirome, 2-32; cefsulodin, 4-32; ceftazidime, 2-16; cefuroxime, 4-16; imipenem, 1-8; meropenem, 1-8; piperacillin-tazobactam, 4/4-64/4). Whereas the BDPhoenix panel NMIC/ID-6 includes both species identification and antimicrobial testing, the VITEK2 card AST-N011 provides susceptibility testing only, so the species name was manually entered into the instrument in order to enable the instrument's expert system to ascertain the β -lactamase phenotype.

3. Results

3.1. ESBL MIC profiles

Thirty-four clinical *E. coli* and *Klebsiella* spp. isolates producing ESBL were grouped on the basis of PCR amplification and sequencing results (Table 1). MICs were determined to obtain the individual resistance profile and were used as reference values when evaluating disagreements observed with the automated systems. Overall, the 34 ESBL strains under study were intermediate or resistant to cefuroxime, the oximino cephalosporins and aztreonam, whereas susceptibility to imipenem (data not shown) and ceftazidime was retained (Table 1). However, the different ESBLs varied in the degree of in vitro resistance they caused. Some, including SHV-5, showed very broad activity and resulted in obvious resistance to all oximino cephalosporins, while others, including SHV-12, SHV-19, and TEM-26, had a 'ceftazidimase' phenotype, with much higher MIC values for ceftazidime than for other oximino cephalosporin drugs. A specific deviation toward cefotaxime ('cefotaximase') was seen in 4 strains containing CTX-M-1. Remarkably, one ESBL strain (EC-9) was also

resistant to ceftazidime, a feature highly unusual for the ESBL enzymes described up to now. Further analysis revealed that EC-9 harbored TEM-52, which was described as a novel type of ESBL capable to hydrolyze the cephamycins (Poyart et al., 1998). It is of note that few strains, most obviously KP-19 and KP-22 harboring SHV-2, demonstrated only marginal ESBL activity, raising only the cefpodoxime MIC to 4 $\mu\text{g}/\text{ml}$ while leaving those of cefotaxime, ceftriaxone, and ceftazidime scarcely above the values seen for nonproducers.

3.2. ESBL detection by automated susceptibility testing systems

All 34 isolates were correctly identified as an ESBL producer by the BDPhoenix system and its BDXpert system. In comparison, by using VITEK2, five isolates out of the same collection of challenge strains were misidentified to have a more susceptible β -lactam resistance phenotype by the Advanced Expert System (Table 3). It was not surprising that the discrepancies were mainly observed with difficult-to-detect strains. In two isolates, namely KP-19 and KP-22, the oximino cephalosporin MICs were so uncharacteristically low (i.e., $\leq 2 \mu\text{g}/\text{ml}$) that it was impossible for the AES to match that pattern to an ESBL phenotype (Table 1). Therefore, any MIC-based system would have underestimated these difficult-to-detect strains. Only cefpodoxime MICs ($> 2 \mu\text{g}/\text{mL}$) would have given correct ESBL detection in these two organisms. On the other hand, given the fact that porin-deficient strains are generally resistant or at least less susceptible to ceftazidime, which was not the case in these strains, it remains obscure why these isolates were categorized as 'impermeability mutants' by the Advanced Expert System. Discrepancies occurred also with the 'ceftazidimase' and 'cefotaximase' strains. These strains conferred clear resistance to either ceftazidime or cefotaxime but had only marginal effects on the MIC of other oximino cephalosporins (Table 1). Although the AES program mentioned that a specific deviation toward a single oximino cephalosporin was highly unusual, one *K. pneumoniae* strain containing 'ceftazidimase' SHV-19 (KP-11) was incorrectly identified as SHV-1 high-level producer, and two strains harboring 'cefotaximase' CTX-M-1 were falsely categorized as K1 hyperproducer (KX-7) or could not be identified (EC-10). The exact reason for the inability of the VITEK2 to detect the resistances expressed by these particular strains remains unknown but an explanation might be that these phenotypes had not been reported when the instrument was developed and thus they were absent in the database.

3.3. K1 detection by automated susceptibility testing systems

K. oxytoca chromosomal β -lactamase (K1) is phenotypically quite similar to ESBL enzymes, and thus difficult to

Table 1
ESBL strains

Tested strains	Broth microdilution MIC ($\mu\text{g/ml}$) ^a							Identified ESBL	Other β -Lactamases ^b	ESBL-Test	
	CAZ	CTX	CRO	ATM	CXM	FOX	CPD			VITEK2	BDPhoenix
<i>Klebsiella pneumoniae</i>											
KP-1	64	2	2	64	4	2	8	SHV-12	SHV-1	+	+
KP-2	4	4	0.03	<0.03	1	2	16	SHV-2a	SHV-1	+	+
KP-3	32	4	4	32	8	2	16	SHV-5	SHV-1	+	+
KP-4	64	4	4	64	8	2	16	SHV-12	SHV-1	+	+
KP-5	>256	16	0.06	0.03	2	4	0.25	SHV-12	SHV-1	+	+
KP-6	32	2	2	32	4	1	4	SHV-12	SHV-1	+	+
KP-7	32	2	2	8	4	1	4	SHV-12	SHV-1, TEM-1	+	+
KP-8	32	2	4	32	4	2	0.06	SHV-12	SHV-1, TEM-1	+	+
KP-9	64	4	4	64	8	2	16	SHV-12	SHV-1, TEM-1	+	+
KP-10	16	4	4	8	64	1	8	SHV-12	SHV-1	+	+
KP-11	128	0.125	0.25	0.125	2	2	1	SHV-19	SHV-1	–	+
KP-12	>256	32	32	>256	64	4	>256	SHV-5	SHV-1	+	+
KP-13	>256	32	8	64	32	2	16	SHV-5	SHV-1	+	+
KP-14	>256	32	8	>256	64	8	32	SHV-5	SHV-1	+	+
KP-15	16	2	2	8	8	1	8	TEM-47	SHV-1, SHV-11	+	+
KP-16	128	4	8	128	8	2	16	SHV-12	SHV-1	+	+
KP-17	32	4	0.03	<0.03	1	2	0.06	SHV-12	SHV-1	+	+
KP-18	8	16	4	2	32	2	16	SHV-2	SHV-1	+	+
KP-19	1	0.5	1	0.5	4	2	4	SHV-2	SHV-1	–	+
KP-20	32	2	4	32	8	2	8	SHV-12	SHV-1, TEM-1	+	+
KP-21	32	4	0.03	0.03	2	2	0.125	SHV-12	SHV-1, TEM-1	+	+
KP-22	2	1	1	0.5	2	2	4	SHV-2	SHV-1	–	+
<i>Klebsiella oxytoca</i>											
KX-4	8	2	2	8	4	1	8	SHV-12	TEM-1	+	+
KX-6	4	64	32	8	>256	1	256	CTX-M-1	ND	+	+
KX-7	1	>256	8	4	>256	1	8	CTX-M-1	ND	–	+
<i>Escherichia coli</i>											
EC-1	2	2	2	16	16	2	16	SHV-2	ND	+	+
EC-2	16	2	4	64	16	2	16	SHV-12	ND	+	+
EC-3	32	4	4	64	16	8	32	SHV-12	ND	+	+
EC-4	4	0.5	0.5	2	4	2	2	SHV-5	ND	+	+
EC-5	16	2	0.06	0.06	8	8	1	SHV-12	ND	+	+
EC-6	2	8	16	4	>256	2	64	CTX-M-1	ND	+	+
EC-9	16	>256	32	16	>256	32	>256	TEM-52	ND	+	+
EC-10	0.5	64	2	0.25	>256	1	8	CTX-M-1	ND	–	+
EC-11	>256	0.5	0.125	4	4	4	2	TEM-26	ND	+	+

^a CAZ, ceftazidime; CTX, cefotaxime; ATM, aztreonam; CRO, ceftriaxone; CXM, cefuroxime; FOX, cefoxitin; CPD, cefpodoxime.

^b ND, not detected.

separate from this mechanism. In order to challenge the automated systems, our panel of testing organisms was expanded by 8 clinical *K. oxytoca* strains with a K1 phenotype (Table 2). The VITEK2 system gave accurate identification in all these strains. In contrast, by the BDPhoenix system and its BDXpert program, respectively, all but one of the K1 strains were falsely flagged as ESBL carriers (Table 3). An explanation might be that in the BDPhoenix system the ESBL testing cascade is triggered by growth in a 1 $\mu\text{g/ml}$ -cefpodoxime screening well. Since MICs for producers of K1 β -lactamase in the studied collection were in part ≥ 2 $\mu\text{g/ml}$ and thus overlapped with the ESBL results, cefpodoxime could not reliably discriminate between both phenotypes. According to our MIC data (Table 2), cefotaxime or ceftazidime would be much better choices to separate the ESBL phenotype from the K1 mechanism

when using the current NCCLS breakpoint (i.e., 2 $\mu\text{g/ml}$). For example, if a ceftazidime MIC of ≥ 2 $\mu\text{g/ml}$ had been used as ESBL indicator, the number of falsely ESBL positive isolates would have been reduced to zero.

4. Discussion

The serious clinical consequences of an ESBL diagnosis create a great need for laboratory testing to accurately identify the presence of these enzymes in clinical isolates. Although detection of ESBL carriage is a problem for traditional microbiologic techniques, it may be even more so for automated susceptibility testing systems. Two different strategies for the identification of this particular resistance phenotype are in common use. In the VITEK2 system,

Table 2
Klebsiella oxytoca K1 strains

Tested strains	Broth microdilution MIC ($\mu\text{g/ml}$) ^a							Identified ESBL	ESBL-Test		
	CAZ	CTX	CRO	ATM	CXM	FOX	CPD		NCCLS ^b	VITEK2	BDPhoenix
KX-1	1	1	4	32	>256	4	4	–	–	–	+
KX-2	0.5	0.25	1	4	32	8	1	–	–	–	–
KX-3	0.06	0.125	1	2	32	2	1	–	–	–	+
KX-5	0.25	0.25	2	16	128	2	2	–	–	–	+
KX-8	0.06	0.25	2	4	64	1	2	–	–	–	+
KX-9	0.125	0.25	2	2	64	1	2	–	–	–	+
KX-10	1	2	4	32	>256	4	4	–	–	–	+
KX-11	1	0.5	4	32	>256	2	2	–	–	–	+

^a CAZ, ceftazidime; CTX, cefotaxime; CRO, ceftriaxone; ATM, aztreonam; CXM, cefuroxime; FOX, ceftioxitin; CPD, cefpodoxime.

^b phenotypic ESBL confirmatory testing according to NCCLS document M100-S12 (NCCLS, 2002).

ESBL prediction is based on the analysis of the MIC pattern to a selection of β -lactam antimicrobials (Sanders et al., 2000). This concept differs from that utilized in the new BDPhoenix system which uses cefpodoxime as a screening drug, followed by testing for synergy between oximino cephalosporins and clavulanic acid. In the present study, we have compared both investigational rapid susceptibility and identification instruments and their expert systems, respectively, in their ability to detect ESBL carriage among a population of clinical *E. coli* and *Klebsiella* spp. with ESBL.

Although the inclusion of a synergy test (cefotaxime-clavulanate and ceftazidime-clavulanate) in the previously developed VITEK system has proven useful to improve

ESBL detection (Sanders et al., 1996), at this time in the VITEK2 system ESBL content is predicted by MIC distribution only (Sanders et al., 2000). Whereas other authors were able to detect even very rarely encountered ESBL variants with this technique (Sanders et al., 2000; Canton et al., 2001), in our hands the MIC-based method failed to identify ESBL carriage for 5 of the 34 ESBL strains tested. In these instances, there was either a strong deviation toward a single oximino cephalosporin in the MIC pattern that made it difficult for the instrument's expert system to match the susceptibility profile to an ESBL phenotype for the species. In this case, an updated and more detailed database could be helpful to improve the performance. Or the MICs of the oximino cephalosporins were so uncharacteristically low that it was impossible for the expert system to match that pattern to an ESBL phenotype. However, it is a well known phenomenon that many strains producing ESBL demonstrate an inoculum effect in that the oximino cephalosporin MICs rise as the inoculum increases (Jorgensen & Ferraro, 2000). Maybe a higher final inoculum density (i.e., 10^7 instead of 10^5 cfu/ml) could have detected this particular cases. Otherwise, the inclusion of a specific ESBL test on the VITEK2 card would be essential.

The false-negative results with the VITEK2 system are serious errors since misidentification of an ESBL strain could lead to incorrect chemotherapy and in consequence be clinically fatal. Considering this correlation, poor discrimination of a K1 phenotype appears to be only a minor error. Nevertheless, misidentification of K1 phenotype is likely to result in implementation of expensive infection control measures in most hospitals, and thus is problematic, too. According to our observations and other sources (Moland et al., 1998; Thomson et al., 1999; Gibb & Crichton, 2000), a better discrimination of the K1 phenotype could be achieved quite easily if the ESBL predicting algorithm would be modified in a manner that places ceftazidime in the screening position of the testing cascade when testing *K. oxytoca*. In this context, it is interesting to note that a 1 $\mu\text{g/ml}$ -ceftazidime well is already available on the BDPhoenix

Table 3
 Strains incorrectly identified by VITEK2 and BDPhoenix

Strain	Resistance mechanism ^a	Assigned phenotype VITEK2/AES ^a	Assigned phenotype BDPhoenix/BDXpert ^a	Cause ^b
ESBL strains				
KP-11	SHV-19	SHV-1 High	ESBL	No phenotype?
KP-19	SHV-2	d_OMP	ESBL	Low-MIC strain
KP-22	SHV-2	d_OMP	ESBL	Low-MIC strain
KX-7	CTX-M-1	K1 High	ESBL	No phenotype?
EC-10	CTX-M-1	unidentifiable	ESBL	No phenotype?
<i>K. oxytoca</i> K1 strains				
KX-1	K1	K1 High	ESBL	Algorithm
KX-3	K1	K1 High	ESBL	Algorithm
KX-5	K1	K1 High	ESBL	Algorithm
KX-8	K1	K1 High	ESBL	Algorithm
KX-9	K1	K1 High	ESBL	Algorithm
KX-10	K1	K1 High	ESBL	Algorithm
KX-11	K1	K1 High	ESBL	Algorithm

^a Abbreviations: K1, *K. oxytoca* chromosomal K1 β -lactamase; d_OMP, deficient in outer membrane protein (i.e., impermeability mutant); High, high-level producer; ESBL, extended-spectrum β -lactamase.

^b Causes for the error in identification included absence of the correct phenotype in the database (no phenotype), peculiarities in the strains itself leading to uncharacteristically low MICs (low-MIC strain), or incorrect design of the underlying ESBL predicting algorithm (algorithm).

panel, but currently its result is processed only after evaluation of growth in the cefpodoxime well.

In summary, our study demonstrated that both fully automated antimicrobial susceptibility testing systems, VITEK2 and BDPhoenix, are reliable tools for detection of ESBL in *E. coli* and *Klebsiella* spp. For VITEK2, the use of an independent ESBL screen might be useful to overcome the weakness to detect low-MIC ESBL strains which is inherent to any MIC-based system. In BDPhoenix, the ESBL predicting algorithm needs a modification to improve discrimination between the K1 and the ESBL phenotype when testing *K. oxytoca* strains.

Acknowledgments

The authors thank BD Diagnostic Systems for kindly supplying the BDPhoenix system and Gram-negative identification and susceptibility panels. We also thank bioMérieux for kindly providing the VITEK2 instrument, and Peter Schäfer and Heimke von Osten for critical reading of the English version of the manuscript.

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