IDENTIFYING RESISTANCE™

International Newsletter
n° 7 • March 2007

BIOMÉRIEUX

Staphylococcus aureus is a major pathogen responsible for a wide spectrum of infections. Since the introduction of methicillin, methicillin-resistant *S. aureus* (MRSA) has spread all over the world and is still one of the leading cause of hospital-acquired infections. More recently, MRSA has also been involved in sporadic community-acquired infections.

MRSA pathogenicity, epidemic spread and resistance to all beta-lactams, as well as to many other drugs, reflects the crucial need for laboratory screening for this organism. However, MRSA detection is still challenging for microbiologists due to low-level resistance expression in some isolates.

Much progress has been made in recent years regarding the diagnosis, either phenotypic (cefoxitin test) or genotypic (specific search of the *mecA* gene in *S. aureus*), the epidemiology (selective chromogenic media for infection controls, molecular typing, *SSCmec* cassette characterisation), and the comprehensive role of toxins in the virulence of MRSA.

It's a great pleasure to see all these aspects revisited in this newsletter by Dr Felten who was the first to foresee the interest of cefoxitin as a surrogate marker of oxacillin for MRSA detection.

Gilles Zambardi

R&D Microbiology – bioMérieux Expert in Antimicrobial Susceptibility Testing

from diagnosis,
the seeds of better health

State-of-the-Art

MRSA revisited

Annie Felten, MD

Annie Felten, MD, is associate professor at the academic hospital Saint Louis in Paris.
She has been working on S. aureus on a routine basis since 1972.
She found the phenotypic marker of methicillin-resistance while looking for the methicillin status of a cluster of pauci-resistant S. aureus isolates from the plastic surgery department in December 1998.
Annie Felten's research interests are antimicrobial resistance, anaerobic infections, and bacterial pathogenicity according to the host status.

Staphylococcus aureus (*S. aureus*) is a major cause of community-acquired (CA) and hospital-acquired (HA) infections and is the primary causative agent of human suppurative skin and deep-seated infections. Much remains to be known about it. According to its plastic genome, it has embarked on many successive pathogenic paths. The introduction in 1959 of anti-staphylococcal semi-synthetic penicillins, oxacillin and methicillin, was followed by the emergence of methicillin-resistant *S. aureus* (MRSA). An additional penicillin-binding protein (PBP), a cell-wall peptidoglycan transpeptidase, named PBP2a - a PBP2 mutant with a low affinity to methicillin - is responsible for methicillin resistance. PBP2a is encoded by the *mecA* gene. This gene is included in a staphylococcal cassette chromosome *mec* (SCC *mec*) which involves genes for the integration and mobility of the *mecA* gene in the bacterial MSSA host *(Hiramatsu)*. It originates by horizontal transfer and recombination from other species, such as ubiquitous *S. sciuri* or

MRSA revisited

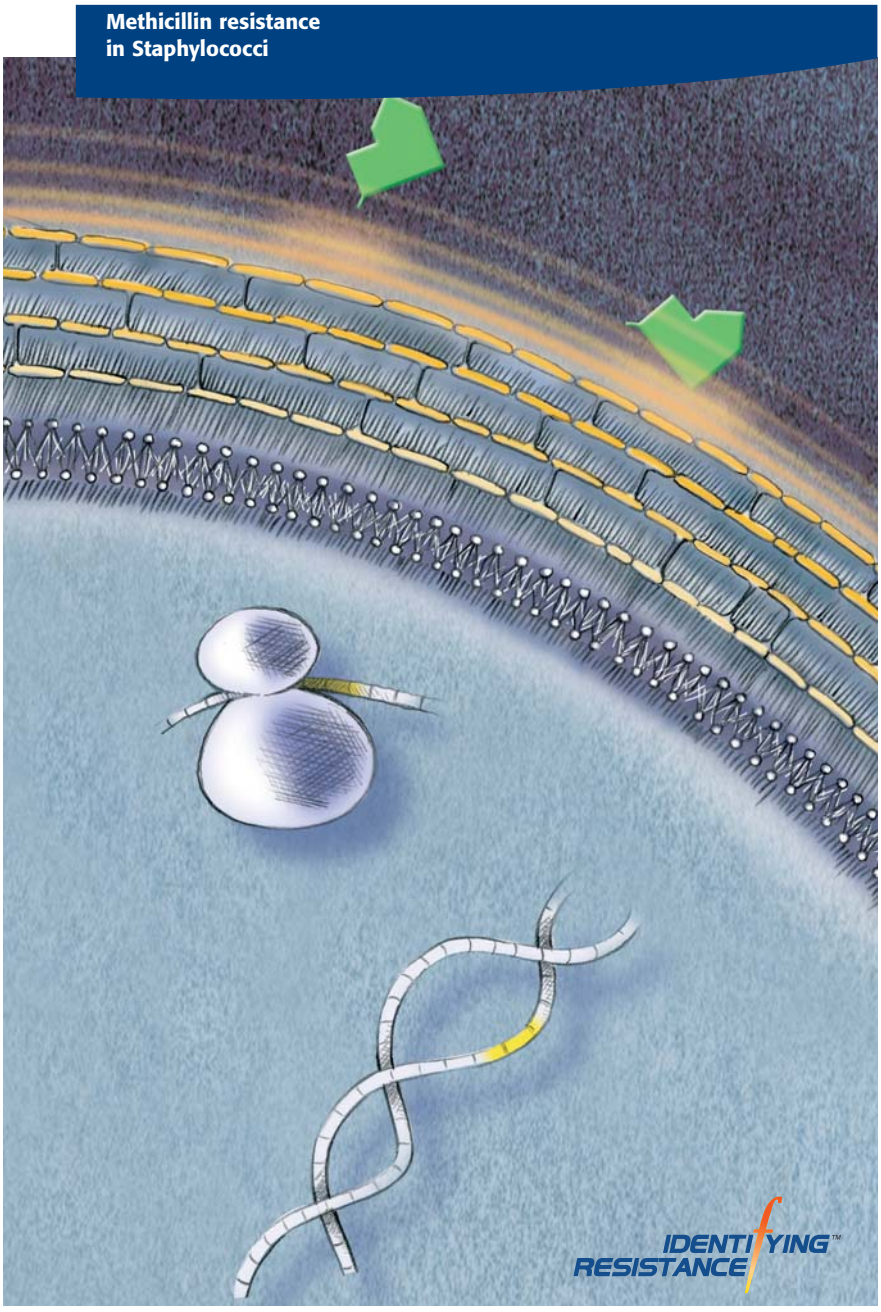

fig. 1

The *S. aureus* chromosome contains the *mecA* gene, inside the SCC*mec* cassette. This gene commands the synthesis of a modified enzyme – PBP2a - building the bacterial cell wall. This modified cell wall makes the strain resistant to all ß-lactams.

commensal *S. epidermidis (Hanssen, Musser)*. Many genes for transcriptional factors regulate the phenotypic expression of methicillin-resistance. MRSAs are resistant to all ß-lactams *in vivo* but may be misdiagnosed as methicilllin-susceptible *S. aureus* (MSSA) by oxacillin tests *in vitro (Felten)*. The reference method to identify staphylococcus methicillin resistance is *mecA* gene detection by gene amplification.

Hospital-acquired MRSA

HA-MRSA were first identified in 1960. They evolved from five major lineages and gave pandemic clones. At first, one multilocus enzyme genotype was predominant *(Musser, Robinson)*. MRSA epidemic clones have arisen from successful epidemic MSSA strains *(Enright)*. The prevalence of MRSA has grown steadily throughout the world to reach 50% in hospitals in Japan and Spain.

The HA-MRSA SCCmec elements belong to type I, II, or III. They carry various resistance genes for a large number of non-ß-lactam antibiotics, aminoglycosides, fluoroquinolones, macrolides, lincosamides, tetracyclines, trimethoprim-sulfonamides, fusidic acid, or rifampin, which allow them to survive selective antibiotic pressures. Despite their growing prevalence in hospitals, MRSAs remained uncommon in the community until the year 2000 *(Chambers)*.

Community-acquired MRSA

MRSA can no longer be regarded merely as a nosocomial pathogen *(Moellering)*. Sporadic reports of CA-MRSA infections first appeared in the 1980s, but 1999 marked the beginning of the current epidemics in Europe, North America, Australia, and New-Zealand *(Vandenesch)*. In the USA, the sentinel event was a series of fatal cases of CA-MRSA infections in children, related to a clone of MRSA widely reported in mid-western USA, MW2 *(Herold)*. The entire genome sequence of MW2 (named USA-400 CA-MRSA by the CDC), was compared with those of two HA-MRSA: N 315 heterogeneous MRSA from Japan and E-MRSA 16 from UK. The USA-400 *mecA* gene was carried by SCCmec type IV, and both HA-MRSA were type II *(Baba)*. USA-400 did not carry any of the multiple antibiotic-resistant genes reported in type II but it carried additional virulence genes. A second clone, multilocus sequence type (ST) ST8 (named USA-300 by the CDC), has since become predominant in the USA *(Chambers)*.

The Panton-Valentine leukocidin (PVL)

PVL is a cytotoxin that causes leukocyte destruction and tissue necrosis. It is associated with primary skin and soft tissue infection *(Boyle)*. The genes coding for PVL are carried on temperate bacteriophages. About 5% of CA-*S. aureus* are PVL-positive, mostly in cases of furonculosis, severe necrotic pneumonia, and cellulitis *(Lina)*. Most are MSSA, but the rate of PVL-positive MRSA is rising. Among these PVL-positive CA-MRSA, many genetic backgrounds are represented in Europe but the clone SCCmec type IVc and ST80 is pandemic *(Holmes)*. In the USA reports, more than 60% of the CA-MRSA USA-400 and USA-300 contain genes encoding for PVL *(Moellering)*. Initial reports include infections among young children, prisoners, homosexual men, competitors in contact sports. Skin infections often have necrotic centers, looking like spider bites *(Diep)*. PVL may be the principal virulence factor responsible for the spread of CA-MRSA or may induce expression of other virulence factors *(Boyle)*. A set of CA-MRSA strains from the USA, Europe, and Oceania was analyzed: most of them shared the SCCmec type IV and the PVL locus, while other toxin-genes were continent-specific *(Vandenesch)*. Isolates were susceptible to most antibiotics, except those from Europe which showed resistance to kanamycin, tetracycline, and fusidic acid. Migration of PVL-positive CA-MRSA in the hospital setting looks likely, and PVL-genes have been found in isolates closely related to the epidemic HA-MRSA clones *(Holmes, Diep, Otter)*.

Detection of methicillin-resistance

Detection of MRSA is made difficult by the presence of subpopulations with heterogeneous expression of methicillin resistance. In very low-level MRSA (class I MRSA) less than 10^{-5} bacteria may express resistance *(Hiramatsu, Felten)*. In the N315 MRSA from Japan, heteroresistance depends on repressor genes, *mecRI* and *mecI*. Low-level and heterogeneous resistance are found in both HA and CA-MRSA. In 5 to 10% of MRSA isolates, methicillin and oxacillin failed to detect MRSA. Staphylococcal oxacillin tests were therefore performed under special conditions: NaCl-enriched medium or 30°C incubation, extended incubation time (48 hrs vs 18 hrs). Nonetheless, some MRSA were misdiagnosed as MSSA. In cases of typical HA-MRSA, the antibiotic resistance pattern led to suspect methicillin-resistance. In cases of CA-MRSA, as they are susceptible to most antistaphylococcal antibiotics and sometimes penicillinase-negative, methicillin-resistance was overlooked *(Blanc, Wannet)*.

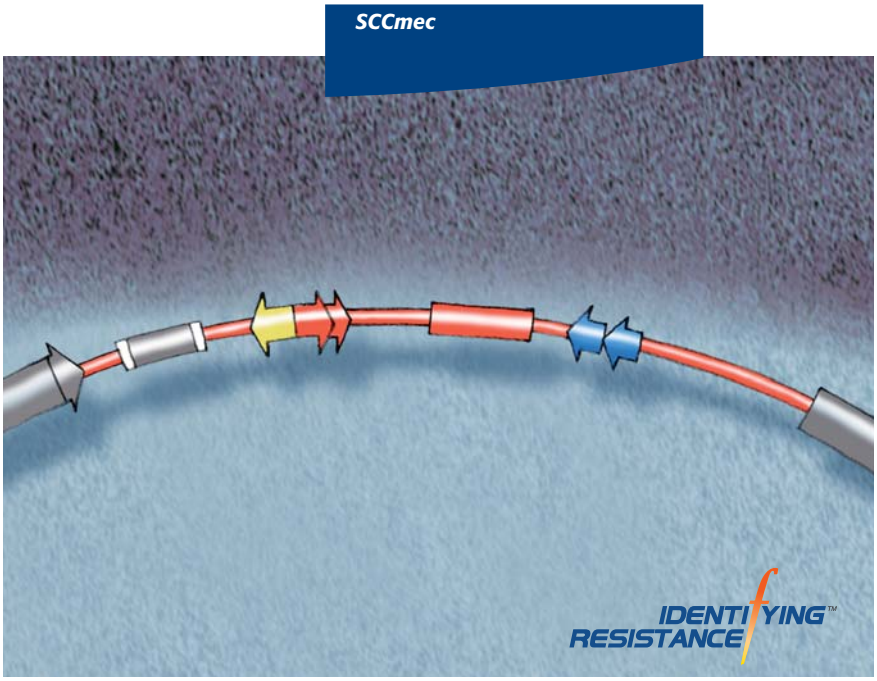

fig. 2

SCCmec
The staphylococcal cassette chromosome (SCC) is a gene cassette common in staphylococci and able to receive the *mecA* gene (yellow), coding for resistance to ß-lactams. It consists of a *mec* gene complex, a *ccr* gene complex, an integration site sequence (IS) and three junkyard regions. Diversity of *mecA* and *ccr* genes defines SCC*mec* types I to VI.

MRSA detection with cefoxitin tests

In 2002, I published a paper describing the suitability of a cefoxitin and moxalactam disk diffusion method for detection of low-level MRSA, which gave 100% sensitivity and 100% specificity respectively in our laboratory *(Felten)*. Soon afterwards, cefoxitin was recognized by others as a good surrogate marker of MRSA, using different methods, disk diffusion on agar, MIC in broth, growth on cefoxitin agar. The cefoxitin disk diffusion test was extended in many countries to detect MRSA, with a specific interpretative diameter or MIC breakpoint according to national technical specifications *(Fernandes, Skov)*. CLSI, as well as CA-SFM, recommends the use of cefoxitin as a marker of MRSA. In 2005, the North American surveillance program on staphyloccccal bloodstream infection compared MRSA detection by oxacillin and cefoxitin disk diffusion and confirmed that the cefoxitin test performed perfectly, while the oxacillin disk gave 6% errors *(Pottumarthy)*. In the case of intermediate inhibition diameters, the latex agglutination test used to detect PBP2a is highly sensitive, especially if performed after induction with cefoxitin *(Bressler, Rohre)*. Cefoxitin MICs corresponding to methicillin-resistance in broth and on solid media are respectively > 4 and ≥ 4 mg/l. To maximize the detection of methicillin-resistance, cefoxitin was coupled with oxacillin in automated instruments, which test staphylococcal antibiotic susceptibility in broth. Results are available more rapidly than by dilution in agar. Cefoxitin media were designed to detect MRSA directly in patients suspected of nosocomial carriage. Selective agar media supplemented with cefoxitin performed better than oxacillin media *(Perry, Smyth)*.

Conclusion

The availability of MRSA detection methods is of primary importance, both in the community and in hospitals. Cefoxitin is a very useful surrogate marker of methicillin-resistance, and the tests are easy to perform. Whenever a *S. aureus* infection needs to be treated by antibiotics, oxacillin is the first choice in MSSA, but is excluded in MRSA even for very-low level MRSA. The cefoxitin tests are highly adapted to the epidemiological detection of human colonization by both HA and CA-MRSA.

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VITEK® 2

MecA Prediction test for Staphylococci with the VITEK 2 System
R. Griffith, A. Doan, S. Messina-Powell, P. Revel, D. Shortridge, M. Ullery
ASM 2005, C-006
The VITEK 2 test compared to mecA PCR gave the following results for S. aureus: 100% positive predictive value, 97.1% negative predictive value, 97.9% sensitivity, and 100% specificity.

Performance evaluation of the GP12 Antimicrobial Investigational Use Only Test Card for Cefoxitin Screen Using the VITEK2 System as Compared to Cefoxitin Disk Diffusion Screen Test and *mecA* PCR
D. Fuller, R. Bruckner, J. Talbot, T. Davis, D. Bruckner, J. Hindler, S. Browon, and M. Traczewski
ICAAC 2006. D-694
This evaluation demonstrates that the performance of the antimicrobial susceptibility test for OXSF on the VITEK2 platform is comparable to conventional testing (Cefoxitin disk diffusion and mecA PCR) in a clinical laboratory.

Berit Riksheim,
from the **Haukeland University Hospital**,
using the VITEK®2 instrument.
This hospital belongs to
the **Bergen Hospital Trust**:
8500 employees,
1100 beds.
67 000+ in-patients,
314 000 out-patients.

Did you know?

Antibiotic classification

Penicillins	Penicillins: **penicillin** Aminopenicillin: **amoxicillin, ampicillin** Ureidopenicillin Carboxypenicillin penicillinase-stable penicillins: **cloxacillin, dicloxacillin, methicillin, nafcillin, oxacillin** Amdinopenicillin
ß-lactams-ß-lactamase inhibitor combinations	**Amoxicillin-clavulanic acid, ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanic acid**
Cephem (parenteral)	Cephalosporin I: **cefazolin, cephalothin, cephapirin, cephradine** Cephalosporin II: **cefamandole, cefonicid, cefuroxime sodium** Cephalosporin III: **cefotaxime, ceftazidime, ceftizoxime, ceftriaxone, cefoperazone** Cephalosporin IV: **cefepime** Cephamycin: **cefmetazole, cefotetan, cefoxitin** Oxacephem: **moxalactam**
Cephems (oral)	Cephalosporin: **cefaclor, cefadroxil, cefdinir, cefditoren, cefetamet, cefixime, cefpodoxime, cefprozil, ceftibuten, cefuroxime axetil, cephalexin, cephradine** Carbacephem: **loracarbef**
Carbapenems	**Ertapenem, imipenem, meropenem**

Did you know?

WEB SITES

www.staphylococcus.net

- ***In-depth description of the SCCmec, and software to search in data bases. Advisory board with top specialists in the Staphylococcus field. Links to www.mlst.net and www.staphylococcus.org (Jutendo University)***

www.cdc.gov/ncidod/hip/dhqp/ar_mrsa

- ***Explanations and data on MRSA.***

Identifying Resistance Symposium

The fifth French VITEK 2 User's meeting was held for two days in June 2006 close to the Lake Annecy in France.
Organized by French Product managers, it gathered together 60 customers or speakers from various parts of France and 15 bioMérieux people mainly from marketing and research departments.

Philippe Dufour, head of R&D Microbiology at bioMérieux-La Balme, and his team introduced and commented novelties in the VITEK 2 system: new tests (cefoxitin, ESBL, ID NH), update of the Advanced Expert System, V4.02 and gave company answers to a users' questionnaire.

External speakers: **Prof. Bonnet**, **Dr Pangon**, **Dr Ros**, **Dr Scheftel** and **Dr Bemer** presented results of studies on antibiotic susceptibility testing, ESBL detection, Imipenem testing with *P. mirabilis*, identification with new ID GN card, as well as a review of recent publications on similar topics.

G. Zambardi, principal scientist at bioMérieux, presented AST-YST, the first automated antifungal test and the new VITEK 2 ESBL test, which, combined with AES provides very quick and accurate results of high value in routine testing for infected patients.
He finished with the latest R&D's idea:
Using the VITEK card format to determine MIC values of a set of ß-lactam antibiotics alone and in combination with specific inhibitors to quickly and accurately identify a wide array of ß-lactam resistance mechanisms.

Did you know?

CLSI (nccls) recommandations

M100-S17 vol.27 January 2007
Adapted from: Table 2C. MIC interpretive Standards (mg/ml) for Staphylococcus spp

- The acronyms **MRSA** or **MRS** (read glossary) are still commonly used even though methicillin is no longer the agent of choice for testing or treatment. *(Comment 1)*
- Testing of oxacillin is preferred (within penicillinase-stable penicillins group) since it is more resistant to degradation in storage, and because it is more likely to detect heteroresistant strains. Results can be applied to the other penicillinase-stable penicillins. *(Comment 9)*
- For oxacillin-susceptible *S. aureus* and CNS, results for parenteral and oral cephems, ß-lactam/ß-lactamase inhibitor combinations, and carbapenems, if tested, should be reported according to the results generated using routine interpretive criteria. *(Comment 2)*
- For oxacillin-resistant *S. aureus* and CNS, other ß-lactam agents, i.e., penicillins, ß-lactam/ß-lactamase inhibitor combinations, cephems, and carbapenems may appear active in vitro but are not effective clinically. Results for these drugs should be reported as resistant or should not be reported. This is because most cases of documented MRS infections have responded poorly to ß-lactam therapy, or because convincing clinical data have yet to be presented that document clinical efficacy for those agents. *(Comment 3)*
- Isolates of staphylococci that are shown to carry the *mecA* gene, or that produce PBP2a, should be reported as oxacillin resistant.
- Isolates that are not shown to carry *mecA* or do not produce PBP 2a should be reported as oxacillin susceptible if oxacillin MICs are < 2 mg/mL.
- Because of the rare occurrence of resistance mechanisms other than *mecA*, isolates that are negative for the *mecA* gene or do not produce PBP2a, but for which MICs are 4 mg/mL, should be reported as resistant. *(Comment 4)*

Oxacillin	S ≤ 2	R ≥ 4	for *S. aureus* and *S. lugdunensis*
	S ≤ 0.25	R ≥ 0.5	for coagulase-negative staphylococci, except *S. lugdunensis*

- Interpretive criteria for CNS correlate with the presence or absence of *mecA* for *S. epidermidis*.
- These interpretive criteria may overcall resistance for other CNS (E.g., *S. saprophyticus*).

For serious infections with CNS other than *S. epidermidis*, testing for *mecA* or the protein PBP2a may be appropriate for strains for which the oxacillin MICs are 0.5 to 2 mg/mL. *(Comment 10)*

Disk diffusion Test for Prediction of *mecA*-mediated Resistance in Staphylococci

Cefoxitin (30 ug)

- *S. aureus* and *S. lugdunensis*.

If zone ≤ 19 mm report Resistant, if zone ≥ 20 mm report Susceptible

- coagulase-negative staphylococci except *S. lugdunensis*

If zone ≤ 24 mm report Resistant, if zone ≥ 25 mm report Susceptible

Glossary

- *S. aureus*: *Staphylococcus aureus*
- CNS: Coagulase-negative staphylococci
- PBP: Penicillin-binding proteins
- MRSA: Methicillin-resistant *Staphylococcus aureus*
- MRS: Methicillin-resistant staphylococci
- SCC: Staphylococcus cassette chromosome

Practical advice

What is the impact of Methicillin resistance?

The prevalence of methicillin resistance is highly variable, ranging from around 1% (north of Europe) to 30% (France) and even 50-60% in some countries (USA, Japan).

Reference and routine methods

Reference method is detection of *mecA* gene or PBP2 detection. Routine methods are phenotypic and best performed using cefoxitin.

Which therapeutic alternative for MRSA infections?

Serious infections should be treated by glycopeptides (vancomycin, teicoplanin) or linezolid.

Why screen for MRSA carriers?

Screening for MRSA carriage on admission to hospital, together with contact isolation of colonized patients, is the most efficient measure for infection control. This strategy is today recommended by official organisms such as the Society for Healthcare Epidemiology of America.[1]
Using systematic screening to control the spread of MRSA is justified on both medical and economic grounds to:

- Reduce the number of MDRO infections by avoiding cross transmission between patients through isolation and auto-infection of colonized patients
- Adjust the antibiotic surgical prophylaxis depending on the patient status
- Optimize the use of isolation beds
- Control the level resistance
- Provide healthcare cost-effectiveness

1. Muto CA, Jernigan JA, Ostrowsky BE, Richet HM, Jarvis WR, Boyce JM, Farr BR. SHEA Guideline for preventing nosocomial transmission of multidrug-resistant strains of Staphylococcus aureus and Enterococcus. Infect Control Hosp Epidemiol. 2003; 24:362-386.

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