

determinant (data not shown). Standard Southern blot analysis using *PvuII*-digested total DNA hybridised with a 680-bp transposase PCR fragment chemiluminescently marked (ECL™ kit; GE Healthcare, Saclay, France) confirmed the absence of hybridisation of the probe (data not shown). The probe was obtained using *Bifidobacterium longum* F8 [4] genomic DNA as a template and the primers TNP-F8F (5'-AGCCAGCCGGAAGTACAACAA-3') and TNP-F8 (5'-GTGGCGGGTGTGTAGGGGCG-3') under the following PCR conditions: 5 min at 95 °C; 30 cycles of 30 s at 94 °C, 30 s at 50 °C and 1 min at 72 °C; and extension time for 5 min at 72 °C.

In conclusion, this study reports the first molecular survey of the tetracycline resistance genes *tet(W)* and *tet(M)* among successive dominant *Bifidobacterium* strains that colonise the human gut. It suggests that distribution of the acquired *tet* genes may be linked to turnover of the dominant *Bifidobacterium* spp. during a period of time for the same individual. Indeed, potential *tet* gene transfer investigation showed no known mobile genetic determinants. This work contributes to the knowledge of *tet* resistance among these human commensal bacteria and gives new insights into the in vivo dynamics of acquired *tet* genes among the intestinal bifidobacteria population.

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Meticillin-resistant *Staphylococcus aureus* blood isolates from the emergency department of a tertiary-care hospital in South Korea

Sir,

With regard to increased community-associated meticillin-resistant *Staphylococcus aureus* (CA-MRSA) infections, the epidemiology of *S. aureus* in the emergency department (ED) becomes important. So far, only a few studies have been performed evaluating MRSA infections among patients in the ED [1–3]. However, there have been few data detailing the prevalence and characteristics of MRSA bacteraemia in the ED, with the exception of a Taiwanese study [2]. Although appropriate empirical antibiotic treatment may influence the outcome of patients with *S. aureus* bacteraemia [2], it is difficult to decide whether vancomycin should be included prior to preliminary microbiology reports owing to the increasing prevalence of CA-MRSA. We prospectively collected *S. aureus* blood isolates from a Korean hospital and categorised them as those from the ED and those from other wards (non-ED). To understand their characteristics, we investigated their antimicrobial resistances and genotypes.

One hundred and twenty-one *S. aureus* isolates were obtained from blood collected from a tertiary-care hospital in Seoul, South Korea, during 2006. Forty-five isolates were obtained from the ED and 76 were from the non-ED. In vitro antimicrobial susceptibility testing was performed using a broth microdilution method according to the Clinical and Laboratory Standards Institute [4]. Multilocus sequence typing (MLST) was performed for all 65 MRSA isolates, as described previously [5]. In addition, *spa* typing was also performed [6]. Staphylococcal cassette chromosome *mec* (SCC*mec*) types of all MRSA isolates were determined using multiplex polymerase chain reaction (PCR) [7]. The Panton–Valentine leukocidin (*pvl*) gene was screened using the PCR method.

Of 121 *S. aureus* isolates, 65 (53.7%) were meticillin (oxacillin)-resistant (Table 1). Fifteen MRSA isolates (33.3%) were identified among the 45 *S. aureus* isolates from the ED, which is significantly fewer than the number of isolates identified from the non-ED (50/76; 65.8%). Moreover, for ciprofloxacin, clindamycin, erythromycin, tetracycline and trimethoprim/sulfamethoxazole (SXT), the antimicrobial resistance rates of isolates from the ED were significantly lower than those from the non-ED (Table 1). MRSA isolates from the ED showed slightly lower resistance rates to all antimicrobial agents relative to those from non-ED, but these differences were not significant.

In MLST analysis, seven sequence types (STs) were identified among the 65 MRSA isolates. Among 15 MRSA isolates from the ED, clonal complex 5 (CC5) (ST5 and ST764) was the most prevalent genotype (7 isolates; 46.7%). Of seven MRSA isolates of CC5, four contained SCC*mec* type II and two were type IV; the other showed a non-typeable SCC*mec* type. Four and three MRSA isolates from the ED belonged to ST239-MRSA-III(A) and ST72-MRSA-IVA, respectively. The remaining MRSA isolate from the ED showed ST20 and a non-typeable SCC*mec* type. Also among 50 MRSA isolates from the non-ED, ST5-MRSA-II was the most predominant clonal complex (22 isolates; 44.0%). As in MRSA from the ED, ST239-MRSA-III(A) was the second most prevalent among those from the non-ED (17 isolates; 34.0%). Eight MRSA isolates from the non-ED showed ST72, of which most showed SCC*mec* type IVA. Two and one MRSA isolates from the non-ED belonged to ST1 and ST30, respectively. No *pvl* genes were detected.

In this study, the first interesting finding is that one-third of *S. aureus* isolates from the ED were MRSA. Although the MRSA rate in the ED was significantly lower than that in the non-ED ($P < 0.001$), MIC₉₀ values (minimum inhibitory concentrations for

Table 1
Antimicrobial resistance of *Staphylococcus aureus* isolates from the emergency department (ED) and from other departments (non-ED).

Antimicrobial agent	Total (n = 121)		ED (n = 45)		Non-ED (n = 76)		P-value
	%R	MIC ₉₀ (mg/L)	%R	MIC ₉₀ (mg/L)	%R	MIC ₉₀ (mg/L)	
Oxacillin	65 (53.7)	>64	15 (33.3)	>64	50 (65.8)	>64	<0.001
Penicillin	116 (95.9)	>64	43 (95.6)	>64	73 (96.1)	>64	0.938
Ciprofloxacin	53 (43.8)	64	12 (26.7)	64	41 (53.9)	64	0.001
Clindamycin	50 (41.3)	>64	12 (26.7)	>64	38 (50.0)	>64	0.006
Erythromycin	72 (59.5)	>64	18 (40.0)	>64	54 (71.1)	>64	<0.001
Tetracycline	52 (43.0)	64	12 (26.7)	>64	40 (52.6)	64	0.003
SXT	22 (18.2)	>32/608	4 (8.9)	0.06/1.18	18 (23.7)	>32/608	0.054
Vancomycin	–	1	–	1	–	1	–

%R, percent resistance; MIC₉₀, minimum inhibitory concentration for 90% of the isolates; SXT, trimethoprim/sulfamethoxazole.

90% of the isolates) did not differ (>64 mg/L in both) (Table 1). The second finding is that antimicrobial resistance rates of MRSA isolates were not significantly different between the ED and non-ED except for SXT. The third finding is that the distribution of genotypes of MRSA isolates also did not differ between the ED and non-ED.

Our results indicated that treatment of serious *S. aureus* infections such as bacteraemia can be challenging. The empirical choice of antimicrobial agents may be problematic in regions with a high prevalence of MRSA. However, considering the time of infection onset after admission to the hospital as well as risk factors associated with acquiring MRSA in hospitals, no true CA-MRSA were identified. In addition, antimicrobial resistance profiles and genotypes of MRSA isolates from the ED did not differ from those of the non-ED, which were shared with those of hospital-acquired (HA)-MRSA in Korea. Thus, MRSA isolates from the blood of patients in the ED must be associated with HA-MRSA. MRSA circulating in Korean hospitals may escape from hospitals and infect humans in the community. In Korea, the border between the hospital and community is now blurring. Features of MRSA isolates in the ED of Korean hospital may be influenced by other factors. In Korea, EDs may sometimes be used as entry points into general wards even for those without the need for immediate medical care. Thus, even patients admitting to the ED may have many opportunities for contact with MRSA colonisers.

Although this study is limited to a single hospital in Korea and therefore the results may not represent all Korean hospitals, most tertiary-care hospitals in large Korean cities may confront similar situations as presented in this study. The present study indicates that clinicians in tertiary-care hospitals should be aware of the high prevalence of MRSA among *S. aureus* in EDs.

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