JOURNAL OF CLINICAL MICROBIOLOGY, Nov. 1995, p. 2842–2846 Vol. 33, No. 11
0095-1137/95/$04.00+0

Vancomycin-Resistant Enterococci Colonizing the Intestinal Tracts of Hospitalized Patients

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Received 27 April 1995/Returned for modification 30 June 1995/Accepted 3 August 1995

A point prevalence culture survey was carried out to investigate the prevalence of fecal carriage of vancomycin-resistant enterococci (VRE) among patients admitted to an 800-bed general hospital where no VRE had been isolated previously. Twenty-two of 636 patients (3.5%) were found to be VRE carriers. Eighteen strains were identified as *Enterococcus faecium*, three were identified as *Enterococcus gallinarum*, and one was identified as *Enterococcus faecalis*. The susceptibilities of the enterococci to ampicillin, vancomycin, and teicoplanin were determined by the disk diffusion and the agar dilution methods. High-level resistance (HLR) to gentamicin and streptomycin was determined by the agar screening method. Eighteen strains (82%) were highly resistant to vancomycin, and four strains (18%) were moderately resistant to vancomycin. Five strains were susceptible to teicoplanin (23%; MICs, ≤8 μg/ml). Only one strain (4.5%, *E. faecium*) showed HLR to gentamicin, and six strains (27%) showed HLR to streptomycin (one *E. faecalis* and five *E. faecium* strains). All 18 *E. faecium* and 1 *E. faecalis* strain carried the *vanA* gene, and 3 *E. gallinarum* strains carried the *vanC* gene. An epidemiological investigation revealed several risk factors for VRE colonization: hospitalization and duration of stay in the hematology department and prior vancomycin treatment. The study demonstrates that the patient's gastrointestinal tract is a possible reservoir for VRE, even in hospitals where VRE infections have not yet been observed. Therefore, we conclude that infection control precautions and restriction of glycopeptide usage may be key issues in limiting the emergence and spread of nosocomial VRE infections.

Although enterococci as such are not particularly virulent, they are becoming more important as nosocomial pathogens (14). This is related to their resistance to several antimicrobial agents, and this resistance can be intrinsic (low-level resistance to penicillin, cephalosporins, and aminoglycosides), as well as acquired (glycopeptides, high concentrations of aminoglycosides). In the United States, the Centers for Disease Control and Prevention recorded a 20-fold increase in the incidence of vancomycin-resistant enterococci (VRE) associated with nosocomial infections between 1989 and 1993 (4). Although VRE have been reported in Europe since 1990 (18), infections caused by these organisms are still extremely rare in Belgium (6, 23). A multicenter study carried out in 1993 (23) indicated that 17% of the *Enterococcus faecium* strains but none of the *Enterococcus faecalis* strains causing infections in hospitalized patients showed resistance to glycopeptides. To our knowledge, no reports on VRE colonization of patients admitted to a hospital where VRE infections have not yet been observed have been published. Therefore, a point prevalence culture survey was carried out to investigate the prevalence of intestinal colonization with VRE among patients admitted to our hospital.

MATERIALS AND METHODS

Prevalence study. In 1993, 25,000 patients were admitted to the St. Jan General Hospital, an 800-bed acute- and chronic-care facility with a university affiliation, for an average of 11 days per admission. The study protocol was approved by the hospital's Ethical Committee. On 22 or 23 November 1993, a rectal swab or fecal sample (as preferred by the patient) was taken from all patients consenting verbally to the study protocol (636 of the 775 patients admitted to the hospital) and was transported to the microbiology laboratory for selective culture of glycopeptide-resistant enterococci.

Selective culture and biochemical identification of VRE. All samples were plated onto two different selective D-Enterococcosel agar plates (bioMérieux, Marcy-l'Etoile, France). The first one was supplemented with 8 μg of vancomycin per ml, and the second one was supplemented with 8 μg of teicoplanin per ml. All plates were incubated aerobically at 35°C for 48 h. From each plate, one or more colonies morphologically resembling enterococci (i.e., dark brown halo) were initially identified by Gram staining, growth in 6.5% NaCl broth, and bile esculin hydrolysis. All presumed enterococci were further identified as described by Facklam and Collins (8) by using the following physiological tests: motility; pyrrolidonyl arylamidase (Rosco Diagnostica, Taestrup, Denmark); leucine aminopeptidase (Rosco Diagnostica); acid formation from mannitol, sorbitol, sorbose, arabinose, raffinose, and sucrose; arginine hydrolysis; and tolerance to tellurite. In parallel, all isolates were also identified by the API-20 S Streptococcus system (bioMérieux), concomitantly with the vancomycin-susceptible reference strain *E. faecalis* ATCC 29212 and two vancomycin-resistant strains *E. faecium* Iowa 1 and *E. faecium* Iowa 2 (11).

PAGE of whole-cell proteins. All strains were grown for 24 h at 37°C on brain heart infusion agar (Difco Laboratories, Detroit, Mich.) and were incubated in a microaerobic atmosphere containing 5% O_2, 10% CO_2, and 85% N_2. Polyacrylamide gel electrophoresis (PAGE) of whole-cell proteins, densitometric analysis, normalization and interpolation of the protein profiles, and numerical analysis were performed as described by Pot et al. (17). All strains were identified by using a database comprising protein patterns for more than 700 strains representing all enterococcal species.

Susceptibility testing. Disk diffusion tests for vancomycin and teicoplanin (Becton Dickinson Microbiology Systems, Cockeysville, Md.) were performed on Mueller-Hinton agar (Becton Dickinson Microbiology Systems) according to the standards of the National Committee for Clinical Laboratory Standards (15). The MICs of vancomycin (Eli Lilly & Co., Indianapolis, Ind.), teicoplanin (Gruppo Lepetit, Milan, Italy), and ampicillin (SmithKline Beecham, Genval, Belgium) were determined by agar dilution on Mueller-Hinton agar with serial twofold dilutions of between 256 and 0.125 μg/ml, and the results were interpreted according to the standards of the National Committee for Clinical Laboratory Standards (16) (for vancomycin, resistance was an MIC of ≥32 μg/ml and susceptibility was an MIC of ≤4 μg/ml; for teicoplanin, resistance was an MIC of ≥32 μg/ml and susceptibility was an MIC of ≤8 μg/ml). The vancomycin-susceptible reference strain *E. faecalis* ATCC 29212 and the two vancomycin-resistant strains *E. faecium* Iowa 1 and Iowa 2 (11) were included in each run.

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TABLE 1. Antimicrobial susceptibilities of 22 VRE

Species	Disk diffusion[a]		MIC (μg/ml)[b]					Genotype by PCR
	Van	Tei	Amp	Van	Tei	Strep	Gen	
E. faecalis	R	R	2	>256	64	>2,000	<500	*vanA*
E. faecium	R	R	32	>256	32	500	<500	*vanA*
E. faecium	R	S	32	>256	32	500	<500	*vanA*
E. faecium	R	I	16	>256	16	<500	<500	*vanA*
E. faecium	R	R	4	>256	16	<500	<500	*vanA*
E. faecium	R	R	128	>256	16	<500	<500	*vanA*
E. faecium	R	R	4	>256	32	500	<500	*vanA*
E. faecium	R	R	32	>256	32	>2,000	<500	*vanA*
E. faecium	R	R	32	>256	32	>2,000	<500	*vanA*
E. faecium	R	R	8	>256	32	500	<500	*vanA*
E. faecium	R	R	32	>256	32	500	<500	*vanA*
E. faecium	R	R	32	>256	32	>2,000	<500	*vanA*
E. faecium	R	R	32	>256	32	500	<500	*vanA*
E. faecium	R	I	32	>256	32	<500	<500	*vanA*
E. faecium	R	I	32	128	8	<500	<500	*vanA*
E. faecium	R	R	128	128	32	>2,000	<500	*vanA*
E. faecium	R	R	4	128	32	500	<500	*vanA*
E. faecium	R	S	32	128	16	<500	<500	*vanA*
E. faecium	R	S	32	8	4	>2,000	<1,000	*vanA*
E. gallinarum	R	S	2	8	0.5	<500	<500	*vanC*
E. gallinarum	I	S	1	8	0.5	<500	<500	*vanC*
E. gallinarum	R	S	1	8	0.5	<500	<500	*vanC*

[a] Van, vancomycin; Tei, teicoplanin; R, resistant; S, susceptible; I, intermediate resistance.
[b] Amp, ampicillin; Van, vancomycin; Tei, teicoplanin; Strep, streptomycin; Gen, gentamicin.

High-level resistance to aminoglycosides was determined by growth of the isolates on four Mueller-Hinton agars containing 500 and 2,000 μg of streptomycin per ml and 500 and 1,000 μg of gentamicin per ml, respectively.

Epidemiological investigation. A case-control study was performed. For each patient colonized with VRE, two patients present in the same ward at the time of study were chosen as controls. The following factors were registered: age, sex, hospital ward at time of sampling, principal diagnosis, length of stay in the hospital during the previous 324 days (i.e., from 1 January 1993 onward), number of days in the hematology or intensive care ward, and antibiotic treatment in the last 324 days (days with any antibiotic and for each group of antibiotics separately). Odds ratio's (ORs) were calculated by using the Epi Info software (7) with 95% confidence limits (Cornfield's approximation as described by Fleiss [9]). Statistical means for cases and controls were compared by the Kruskal-Wallis one-way analysis of variance (19).

Amplification of *vanA*, *vanB*, and *vanC* genes by PCR. For amplification of the *vanA*, *vanB*, and *vanC* genes, the oligonucleotide primers published by Clark et al. (5) were chosen. The PCR mixture was slightly modified. All reactions were performed in a 50-μl volume. Briefly, between 5 and 10 bacterial colonies from a blood agar plate incubated overnight were suspended in the reaction mixture, which contained 10 mM Tris HCl (pH 8.3), 50 mM KCl, 1.5 mM $MgCl_2$, 200 μM (each) dATP, dCTP, dGTP, and dTTP, 0.1 μM (each) primer, and 1 U of *Taq* polymerase (Goldstar; Eurogentec, Seraing, Belgium). A Perkin-Elmer Cetus model 9600 DNA Thermocycler was used and was programmed as follows: lysis and denaturation for 30 s at 94°C, hybridization for 30 s at 58°C, and elongation for 30 s at 72°C for 30 cycles; the first lysis and denaturation step was for 10 min at 95°C, and the last elongation step was for 10 min at 72°C. All glycopeptide-resistant enterococci and 10 glycopeptide-susceptible control strains were analyzed for the *vanA*, *vanB*, and *vanC* resistance genes. The reference strains included were *E. faecium* Iowa 1 (carrying the *vanA* gene) and *E. faecium* Iowa 2 (carrying the *vanB* gene) (11). The amplicon was revealed by agarose gel electrophoresis on a 2.0% agarose (Hispanagar, Burgos, Spain) gel prepared in 0.5× TBE (Tris-borate-EDTA) buffer and was stained with ethidium bromide. Amplicons were visualized by using UV transillumination.

RESULTS

Prevalence of VRE colonization. Six hundred thirty-six of the 775 hospitalized patients (82.1%) agreed to participate in the study. *Enterococcus*-like organisms were obtained from 135 of the patients (21.2%), with a dark brown coloration of the medium. The isolates grew massively on at least one selective medium. However, 113 (83.7%) of these isolates were identified as *Leuconostoc* spp. or vancomycin- and teicoplanin-susceptible enterococci. Twenty-two patients (3.5%) carried enterococci with decreased susceptibility to vancomycin (MICs, >4 μg/ml). Isolates from 17 patients (2.7%) also showed decreased susceptibility to teicoplanin (MICs, >8 μg/ml), and 5 isolates were teicoplanin susceptible (MICs, ≤8 μg/ml).

Identification. All 22 strains grew at 45°C and in 6.5% NaCl and were pyrrolidonyl arylamidase and leucine aminopeptidase positive. None of the strains was pigmented. Eighteen of the 22 strains were identified as *E. faecium*, 1 was identified as *E. faecalis*, and 2 were identified as *Enterococcus gallinarum*; 1 of the last two strains was presumed to be *E. gallinarum*, although it did not ferment raffinose. The API-20 S Streptococcus system misidentified the three *E. gallinarum* strains as *E. faecium* but correctly identified all other isolates. The identification of the 22 strains was confirmed by comparative analysis of their whole-cell protein profiles by PAGE (data not shown).

Susceptibility to antimicrobial agents. Table 1 provides the results of the in vitro susceptibility tests. By the disk diffusion method, 21 of the 22 strains were resistant to vancomycin (zone diameter, ≤14 mm) and one strain showed an intermediate resistant result (zone diameter, <17 mm but >14 mm). Thirteen strains were also resistant to teicoplanin (12 *E. faecium* strains and 1 *E. faecalis* strain), 3 strains showed intermediate resistance, and 6 strains were susceptible. Determination of the MIC by the agar dilution test confirmed decreased susceptibility to vancomycin for all 22 strains. Twelve *E. faecium* and one *E. faecalis* strains were resistant to vancomycin and teicoplanin (MICs, ≥32 μg/ml). Four *E. faecium* isolates were resistant to vancomycin and had intermediate resistance to teicoplanin (MICs, 16 μg/ml). All three *E. gallinarum* strains and one *E. faecium* strain had intermediate resistance to vancomycin (MICs, 8 μg/ml) and were susceptible to teicoplanin (MICs, 0.5 and 4 μg/ml for the *E. gallinarum* strains and the *E. faecium* strain, respectively). Finally, one *E. faecium* strain was resistant to vancomycin (MIC, 128 μg/ml) and susceptible to teicoplanin (MIC, 8 μg/ml). Eight enterococci remained susceptible to ampicillin (MICs, <16 μg/ml). Only 1 of the 22 enterococci showed high-level resistance to gentamicin (MIC,

TABLE 2. Epidemiological data for patients colonized with VRE

Sex[a]	Age (yr)	Department[b]	Diagnosis	Isolate	Survived	Stay (days) in different hospital wards in the last 324 days[b]								Previous antibiotic treatment (days) in the last 324 days[c]					
						SUR	ICU	HEM	INT	GYN	PED	Chr	Total	Pen	Cef	Gly	Fq	Ag	Total
F	81	CHR	Breast carcinoma	497	Yes							71	71				10		10
F	36	GYN	Cesarean delivery	615	Yes					6			6						
F	54	HEM	Acute leukemia	175	Yes			92					92	33	28	22	52	76	249
M	72	HEM	Non-Hodgkin's lymphoma	27	Yes			34	23				57	8	3	10	3	3	49
F	79	HEM	Non-Hodgkin's lymphoma	9	No			65					65	15			9	13	38
F	69	HEM	Diabetes, renal impairment	11	Yes		2	96	13				111	6		2		2	10
M	81	ICU	Abdominal aneurysm	90	No		13						13	3	6				15
M	44	INT	Stroke	126	Yes				67				67	23				1	24
F	68	INT	Pulmonary embolism	115	Yes				29				29	9			4		12
M	81	INT	Contusio cerebri	360	Yes		6		11				17	2	6				27
F	61	INT	Cholecystitis	323	Yes	7			2				9	9	1			7	17
M	71	INT	Duodenal angioma	325	Yes				16				16						
F	0	PED	Hydrocephalia with shunt	398	Yes						49		49	1	1				5
F	62	SUR	Plastic surgery	302	Yes	19							19						
F	81	SUR	Shoulder prosthesis	162	Yes	21							21	10	1				11
M	38	SUR	Spermatocoele	340	Yes	1							1						
M	82	SUR	Trigeminus neuralgia	208	Yes	1			53				54						
F	84	SUR	Total hip replacement	565	Yes	18							18		5				9
M	94	SUR	Hematoma cerebri	74	Yes	9	3		1				13		1		5		6
M	68	SUR	Hematoma cerebri	66	Yes	19	54						73	11	15	45	15	15	129
F	51	SUR	Hematoma cerebri	80	Yes	5	43						48	40			10	8	63
M	72	SUR	Pulmonary carcinoma	406	No	1							1						

[a] F, female; M, male.
[b] SUR, surgery; ICU, intensive care; HEM, hematology; INT, internal medicine; GYN, gynecology; PED, pediatrics; CHR, chronic care.
[c] Pen, all penicillins; Cef, all cephalosporins; Gly, glycopeptides; Fq, fluoroquinolones; Ag, aminoglycosides.

>1,000 μg/ml), and 6 of them showed high-level resistance to streptomycin (MICs, >2,000 μg/ml).

Mechanisms of resistance. PCR demonstrated that all 18 *E. faecium* strains and 1 *E. faecalis* strain carried the *vanA* gene. However, two *E. faecium* isolates were phenotypically not classified as *vanA* according to the criteria published by Arthur and Courvalin (1). Isolate 175 had low-level resistance to vancomycin and was susceptible to teicoplanin, and isolate 11 was borderline susceptible to teicoplanin (MIC, 8 μg/ml). All three *E. gallinarum* strains were phenotypically classified as *vanC*, and the *vanC* gene was detected by PCR.

Distribution of VRE carriers in the hospital. Table 2 provides information on the 22 VRE carriers encountered in the study. Their distribution throughout the hospital was not homogeneous. Nine of the 22 carriers were situated in the surgical wards, but the prevalence in this department was only 4.9%. Four patients were admitted to the hematology ward, where the prevalence was 10.8%, more than double in comparison with that on other wards ($P < 0.05$). Carriers were also identified, although in small numbers, in several departments considered less at risk for nosocomial transmission (maternity ward, 1 carrier among 34 patients; chronic care, 1 carrier among 125 patients; pediatrics, 1 carrier among 62 patients). Carriers were not found more frequently in the intensive care ward than in the rest of the hospital (5.4%; $P > 0.1$).

Risk factors for VRE colonization. An overview of the epidemiologic data for the VRE carriers and for the subjects in the case-control study is provided in Tables 2 and 3. Sex, mean age, and survival rates were comparable in both groups. The mean duration of hospital stay for VRE carriers was more than 38 days. This is significantly longer ($P < 0.05$) than the average of 13 days for all patients admitted in 1993. The mean numbers of antibiotic treatment days in the 324 days prior to the study were 22.1 and 30.2 days in the control patients and the VRE carriers, respectively ($P > 0.1$). Carriers had received the following drug classes more often than controls: glycopeptides (OR, 1.73), aminoglycosides (OR, 2.22), and antibiotics all together (OR, 1.85). The differences were not statistically significant ($P > 0.1$), however. Because VRE carriers were significantly more frequently found in the hematology ward than in other departments (10.8 versus 3.1%, respectively; $P < 0.05$), we investigated the relation of risk factors between cases and controls from this department separately (Table 3). No difference was found regarding sex, mean age, or survival rate. However, the mean duration of stay was considerably longer for cases than for controls (72 versus 34 days, respectively; $P = 0.06$). Each of the four cases had received antibiotics in the 324 days prior to the study, but so had 56% of the controls. However, ORs of 18 and 10, respectively, suggested that VRE carriers had received glycopeptides ($P = 0.08$) or quinolones ($P = 0.11$) more often, although the differences were statistically not significant. Carriers tended to have received antibiotic treatment longer than controls (mean of 87 versus 41 days, respectively, $P = 0.09$), and the difference in the duration of glycopeptide treatment was important, but it also was not statistically significant (mean, 8.5 versus 5.7 days, respectively; $P = 0.12$).

DISCUSSION

Glycopeptide-resistant enterococci have become a major threat to hospitalized patients. Like methicillin-resistant *Staphylococcus aureus*, VRE can cause important nosocomial epidemics and can, increase morbidity, mortality, and costs related to admission to the hospital. The presence of VRE in clinical samples in Belgium is still very low, and no VRE epidemics have been described. In the St. Jan General Hospital of Bruges, no infection caused by VRE was ever diagnosed. Little is known about the epidemiology of VRE colonization outside of the hospital environment. The investigations of

TABLE 3. Comparison of risk factors between 22 patients colonized with VRE and 44 controls

Characteristic	All departments				Hematology department			
	Cases	Controls	OR	*P* value	Cases	Controls	OR	*P* value
No. of patients	22	44			4	7		
Male (no. [%])	10 (45)	25 (57)		>0.1	1 (25)	5 (71)		
Mean age (yr)	65	61		>0.1	68.5	64		>0.1
Survived (no. [%])	19 (86)	39 (89)	0.81	>0.1	3 (75)	6 (86)		>0.1
Mean duration of stay (days)	38.6	43.0		>0.1	71.8	34.4		0.06
Mean antibiotic therapy (days)	30.2	22.1			86.5	40.9		0.09
No. (%) of patients who had previously received:								
Glycopeptides	4 (18)	5 (11)	1.73	>0.1	3 (75)	1 (14)	18	0.08
Aminoglycosides	8 (36)	9 (21)	2.22	>0.1	4 (100)	2 (29)		0.05
Fluoroquinolones	8 (36)	14 (32)	1.22	>0.1	3 (75)	2 (29)	10.5	0.11
Any antibiotic	16 (73)	26 (59)	1.85	>0.1	4 (100)	5 (56)		>0.1
Mean glycopeptide therapy (days)					8.5	5.7		0.12

Torres et al. (22) and Jordens et al. (12) have suggested that VRE can be part of the intestinal microflora of patients inside and outside of the hospital. The latter investigators also demonstrated vancomycin-resistant enterococci from animal reservoirs (2). However, those studies investigated colonization in areas where nosocomial VRE infections and epidemics were ongoing. Therefore, contamination of the environment from the hospital could not be excluded. The present study demonstrates that about 1 of every 29 patients in St. Jan General Hospital may be colonized with VRE. Detection of VRE in patients without signs of infection or without evidence of a hospital epidemic may suggest that the nosocomial spread is secondary to the emergence of VRE, possibly prior to hospital admission, and selection from the intestinal flora of the patients after admission to the hospital. Moreover, since two patients in the present study had been in the hospital for only 1 day before VRE were detected, we suspect that VRE colonization can be acquired in the community outside the hospital environment, as suggested by Torres et al. (22) and Jordens et al. (12).

The present study detected not only vancomycin-resistant *E. faecium* but also *E. faecalis* and *E. gallinarum*. The question may be asked why vancomycin-resistant *E. faecalis* isolates are only rarely encountered in patients with nosocomial infections even though they are present in the human enteric tract.

Our study also confirms the difficulty in identifying glycopeptide resistance in *Enterococcus* spp. by the disk diffusion method, as previously mentioned by Tenover et al. (21) and Swenson et al. (20), since it failed to detect 1 of 13 teicoplanin-resistant enterococci. The results of PCR indicate that the phenotypic appearance represented by the susceptibility patterns can also be unreliable in identifying the presence of *vanA*, *vanB*, or *vanC* genes. Six of 19 *E. faecium* isolates (31.6%) with *vanA* genotypes would have been missed on the basis of the results of the disk diffusion method, and MICs for 2 isolates (10.5%) were beyond the accepted range for isolates of the *vanA* phenotype (1). Future prevalence culture surveys must also take into account the fact that both *Leuconostoc* spp. and glycopeptide-susceptible enterococci can be mistaken for VRE on the basis of growth and colony morphology on culture medium containing 8 μg of vancomycin per ml. Identification of enterococci with the API-20 S Streptococcus system appears to be unreliable since this system misidentified all three *E. gallinarum* isolates. It is therefore imperative to perform standardized reference methods for identification, susceptibility testing, and PCR in all epidemiological studies on VRE.

As to the epidemiology of VRE colonization, certain risk factors reviewed by Korten and Murray (13) also seem to be involved in the present study. Colonized patients had been admitted to the hospital longer than the average patient, but such times were not significantly different from those for VRE-free control patients hospitalized in the same wards. Therefore, we conclude that VRE colonization is more frequently found in wards where patients tend to stay longer. The relation between VRE colonization and previous antimicrobial therapy and its duration, investigated for all 22 cases and controls, is not obvious. The mean number of antibiotic treatment days was high, and determination of the OR suggested a relation with the kinds of antibiotics administered, especially for glycopeptides and aminoglycosides. However, the differences between controls and cases were not statistically significant and do not confirm the relation with risk factors reported during nosocomial VRE epidemics in other hospitals (3, 10) except for patients in the hematology ward. This may be due to the limited number of cases and controls in our study, but it may also indicate that patients colonized with VRE represent population epidemiologically different from patients who acquire VRE during a nosocomial outbreak. However, a relation between the discovery of VRE and admission on the hematology ward was demonstrated; VRE carriers were significantly more frequently found on this ward ($P < 0.05$) and tended to have stayed there longer than control patients, although this difference was not statistically significant. In this ward, there was a trend for VRE-colonized patients to have received glycopeptides and aminoglycosides, and possibly fluoroquinolones, more often than controls (but the differences not statistically significant). A difference in the duration of glycopeptide therapy was detected, although this difference was not statistically significant. On the basis of the conclusions presented above, we believe that the use of antibiotics, in particular, glycopeptides,

should probably be dramatically restricted in order to avoid the selection of VRE, which are already part of the human microflora.

Our institution maintains a policy of nursing patients colonized with multiresistant bacteria (e.g., methicillin-resistant *S. aureus* and cefotaxime-resistant *Klebsiella pneumoniae*) in isolation. However, since the present study indicates that at least 3.5% of hospitalized patients are healthy VRE carriers, we conclude that the spread of nosocomial VRE epidemics probably cannot be prevented only by nursing VRE-infected patients in isolation. Preferentially, compliance with basic hygienic measures for all patient care, like systematic hand disinfection, wearing gloves, and taking universal precautions when handling blood and body fluid, should be increased.

ACKNOWLEDGMENTS

P.V. is indebted to the National Fund for Scientific Research (Belgium) for a position as a postdoctoral research fellow. This work was partially supported by a grant from Merrell Dow Belgium.

We thank Kathy Claeys and Hilde Jannes for logistic support during the study, Annemie Lambert and Jean-Marie Fossepré for technical assistance in the laboratory, and B. Pot (University of Ghent) for permission to use the *Enterococcus* reference database for comparison of whole-cell protein electrophoresis. The authors are grateful to R. N. Jones (Iowa City) for the reference strains *E. faecium* Iowa 1 and Iowa 2.

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