

# Prevalence of colonisation and resistance patterns of vancomycin-resistant enterococci in healthy, non-hospitalised persons in Switzerland

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## Summary

In order to obtain data about the prevalence and the resistance profiles of vancomycin-resistant enterococci (VRE) in the community, we investigated fecal samples of employees (n = 1026) from food processing companies of different Swiss cantons. The susceptibility of the isolated VRE-strains against penicillin, ampicillin, tetracyclin, ciprofloxacin, chloramphenicol, streptomycin and gentamicin as well as the minimal inhibitory concentrations (MIC) for vancomycin and teicoplanin were determined by agar diffusion and microbroth dilution, respectively. The type of vancomycin-re-

sistance (*vanA*, *vanB*, *vanC*<sub>1</sub>, -C<sub>2</sub> or -C<sub>3</sub>) was confirmed by polymerase chain reaction (PCR).

A ratio of 50 samples out of 1026 (4.9%) was found VRE-positive, all of the isolated strains carried the *vanA*-resistance gene. Compared to recent publications from other European countries, these results represent a rather high prevalence of VRE-carriers in the community.

*Keywords:* vancomycin-resistant enterococci; prevalence; colonisation

## Introduction

Enterococci are often responsible for nosocomial infections such as urinary tract infections, endocarditis or bacteremia [1]. Resistance of these microorganisms against the first choice therapy (aminopenicillin/aminoglycosides), requires alternative treatment with the glycopeptides vancomycin or teicoplanin. In the late 1980's, enterococci developed resistances even against these antibiotics and were called vancomycin-resistant enterococci (VRE).

In the USA, this kind of resistance was traced back to an increased use of glycopeptide antibiotics in human medicine mainly by oral administration [2, 3], while the origin of VRE in Europe was at-

tributed to an extended use of the glycopeptide-antibiotic avoparcin as a growth promoter for pigs and poultry and the following transmission of cross-resistant strains to humans [4]. Hence, a feeding prohibition of glycopeptide-antibiotics in veterinary medicine was imposed in Europe in 1996/97.

The authors' interest in the present study was to investigate the proportion of VRE-carriers in the community because they could act as an outbreak-source, e.g. when they are admitted to hospital and subjected to the selective pressure of antibiotics on the gut flora [2, 5].

## Materials and methods

Between October 1998 and November 1999, 1026 fecal samples of employees from food industries in different Swiss cantons, which were received for an annual *Salmonella*-monitoring, were also tested for the presence of vancomycin-resistant enterococci. A single stool specimen was obtained from each subject. The involved persons belonged to an urban or suburban population.

After enrichment of the samples at 45 °C in buffered pepton-water (Oxoid) in an overnight culture, a bile-esculin-acid-agar (Becton Dickinson), supplemented with 16 µg/ml vancomycin (Sigma), was inoculated with the enrichment broth (37 °C/48 h). The high specificity (89.3%) and sensitivity (92.7%) of 16 µg/ml vancomycin for VRE screening was shown previously [6]. One suspect colony

per sample was subcultivated on sheep-blood agar (Difco) and identified by Gram-staining, catalase-reaction, growth at 45°C and in 6.5% sodium chloride. The differentiation was performed using API 20 Strep and API 50 CH (BioMérieux).

Antibiotic susceptibility of the isolated strains was tested by agar diffusion [7] on Mueller-Hinton-Agar (Oxoid; disks: BioMérieux) towards penicillin (10 units), ampicillin (10 µg), tetracyclin (30 µg), ciprofloxacin (5 µg), chloramphenicol (30 µg), streptomycin (10 and 300 µg)

and gentamicin (10 and 120 µg). The minimal inhibitory concentration (MIC) of vancomycin (Sigma) and teicoplanin (Aventis) was determined by microbroth dilution [8] in Mueller-Hinton-Broth (Becton Dickinson). Interpretation was done in accordance to the criteria of the NCCLS [9].

The determination of the glycopeptide resistance genotype (*vanA*, *vanB*, *vanC*<sub>1</sub>, -C<sub>2</sub> or -C<sub>3</sub>) by PCR was performed as described previously [10].

## Results and discussion

A uniform growth of suspect colonies on vancomycin-supplemented agar was found in 63 samples. Differentiation and identification revealed 13 isolates as *Pediococcus pentosaceus*, an intrinsically vancomycin-resistant Gram positive organism. *Pediococcus* species can exceptionally act as opportunistic pathogens, but antibiotic resistance does not pose a problem [11].

The majority of the remaining 50 isolates were *E. faecium* (47 strains), one was *E. faecalis* and two strains were identified as *E. durans*. Resistance patterns against the tested antibiotics are shown in

Table 1. MIC's of vancomycin and teicoplanin ranged from 512 to 1024 µg/ml and from 32 to 1024 µg/ml, respectively. All isolates carried the *vanA*-resistance-gene.

Thus, resulting prevalence of VRE-carriers sums up to 4.9%, most of them represented by *E. faecium*, the second most frequently identified species in enterococcal infections. Since species identification is done by phenotypical methods, the true distribution of the species might be different from our results [12]. However, all VRE have an acquired type of resistance (*vanA*), which

**Table 1**  
Resistance patterns  
in the 50 VRE strains.

No. of resistances <sup>1</sup>	no. of strains	resistance or intermediate susceptibility to:
1	1	TE
	1	S
2	1	P, TE
	1	P, S
	1	TE, CIP
	1	GM, S
3	1	TE, C
	4	P, TE, S
	2	TE, GM, S
	1	TE, GM, S <sup>2</sup>
	1	P, GM, S
	1	TE, CIP, S
4	7	P, TE, GM, S
	1	P, TE, GM, S <sup>2</sup>
	1	P, TE, GM <sup>2</sup> , S <sup>2</sup>
	4	P, TE, CIP, S
	2	P, TE, CIP, S <sup>2</sup>
	4	TE, CIP, GM, S
	1	TE, CIP, GM, S <sup>2</sup>
	1	P, TE, C, S
	1	P, AM, TE, S
5	7	P, TE, CIP, GM, S
	1	P, TE, CIP, GM, S <sup>2</sup>
	1	P, TE, CIP, C, S
	1	P, AM, TE, C, S
6	2	P, TE, CIP, C, GM, S

<sup>1</sup> Beyond the glycopeptide-resistance

<sup>2</sup> High level aminoglycoside-resistance

P: penicillin; AM: aminopenicillin; TE: tetracyclin; CIP: ciprofloxacin; C: chloramphenicol; GM: gentamicin; S: streptomycin.

is transferable to other enterococci and associated with nosocomial epidemics [13]. Furthermore, the resistance patterns show, that for three out of 50 VRE-carriers, neither the therapy of choice (ampicillin/aminoglycoside) nor the alternative treatment (glycopeptide) would be effective in case of infection. Nevertheless, despite the significant proportion of VRE-carriers in the community, resistance in enterococci still plays a minor role in infections in Switzerland.

Assuming that the prevalence of VRE-carriers in the community is caused by the use of avoparcin in veterinary medicine, one would expect a decrease of the prevalence after the feeding ban of this antibiotic. This has been observed for instance in Germany, where 12% VRE-carriers were found among 100 non-hospitalised humans from a country-like region in 1994 [14] whereas at the end of 1997, nearly 2 years after discontinuation of avoparcin in animal husbandry, only 3% were de-

tected in a group of 400 non-hospitalised persons from the same area [15]. In 1998, 0.95% of a group of 210 German students with an urban background were shown to be VRE-positive [16]. In France, in 1997, Gambarotto et al. revealed 1.8% VRE-carriers in a group of 169 persons from a cattle rearing area [17]. In comparison to the cited recent studies in Germany and France, we detected a rather high prevalence of VRE-carriers in our study group. Since older data are lacking, a temporal trend cannot be documented for Switzerland.

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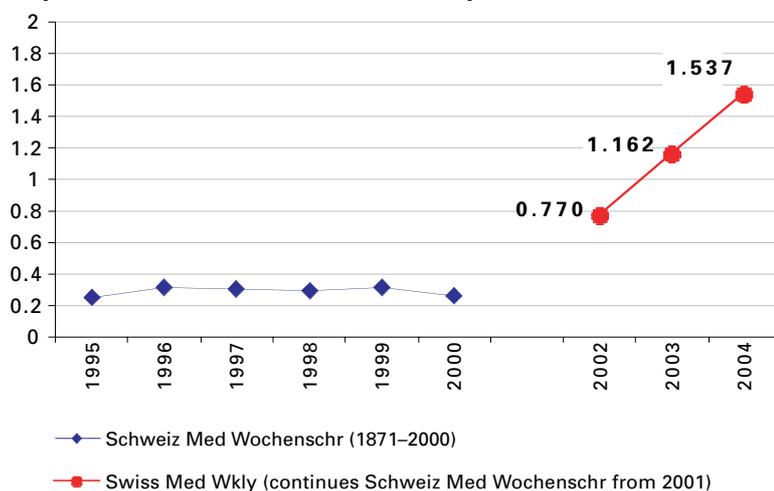
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