Presence of Vancomycin-Resistant Enterococci in Farm and Pet Animals

LUC A. DEVRIESE, 1,2 MARGARET A. IEVEN, 1 HERMAN GOOSSENS, 1 PETER VANDAMME, 1,2 BRUNO POT, 1 JOZEF HOMMEZ, 1 and FREDDY HAEBROUCK 1

Faculty of Veterinary Medicine, University of Ghent, B-9820 Merelbeke, 1 Department of Clinical Microbiology, Antwerp University Hospital, B-2650 Edegem, 2 Faculty of Sciences, University of Ghent, and BCCM/LMG Culture Collection, 3 B-9000 Ghent, and Regional Veterinary Laboratory, B-8820 Torhout, 4 Belgium

Received 16 February 1996; Returned for modification 30 May 1996; Accepted 18 July 1996

Enterococcus faecium strains with vanA-mediated glycopeptide resistance were isolated by enrichment culture from the intestines and feces of several animal species, mainly horses and dogs (8% positive), chickens (7% positive), and pigs (6% positive). Other vanA-positive enterococcal strains were identified as E. durans in gallinaceous birds, E. faecis in a horse, and E. gallinarum in a pheasant. Samples from pigeons, cage birds, and ruminants were negative. It was concluded that vancomycin resistance is widespread among isolates from farm and pet animals.

Vancomycin (glycopeptide)-resistant enterococci (VRE) may cause serious problems in hospitalized patients. Until recently, the possible presence of such strains in animals was unknown and remained uninvestigated, probably because glycopeptide antibiotics are not used therapeutically in veterinary medicine. In Europe, one member of this antibiotic group, vanA-carrying vancomycin-resistant Enterococcus faecium strains are cross-resistant to avoparcin (12). In a report on the in vitro susceptibility of animal enterococci isolated in 1979 to growth-promoting antibiotics (7), all strains investigated were found to be susceptible to avoparcin. In recent years, however, with the finding of VRE in wastewater of sewage treatment plants (2, 13, 16), feces of farm animals (1, 2, 12), meat (2, 11, 12), feces of nonhospitalized persons in Europe and the United Kingdom (10, 17), and in hospitals without vancomycin-resistant infections (8), interest in the possible role of animals as a source of vancomycin-resistant strains increased. In investigations carried out in Germany (11, 12) and Denmark (1), the occurrence of vancomycin resistance was linked to the use of avoparcin as a growth-enhancing antibiotic in animals.

In view of a possible involvement of animals in the spread of VRE, we undertook a broad survey of the presence of such strains in farm and pet animals.

MATERIALS AND METHODS

Samples. The samples examined were collected in 1995 from animals originating from 557 different farms or owners in Belgium (Table 1). Fecal samples from 63 pigeons, 30 horses, 12 rabbits, 6 parrots, and 3 pigeons, litter samples from 35 poultry farms, and anal swabs from 14 cats and 9 dogs were cultured within 1 or 2 days after collection. The remaining samples were taken from the intestines of animals sent in for postmortem diagnosis. These were stored for up to 3 weeks at −30°C before examination. Except for the poultry litter, all samples were collected from individual animals, and only samples from one animal per farm or owner were examined.

Enrichment culture of VRE. Samples of 0.5 to 1 g were homogenized in 5 or 10 ml of kanamycin tryptic soy broth (Lab M, Bury, United Kingdom) supplemented with 20 μg of vancomycin per ml, as described by Bates et al. (2). Tubes whose contents turned turbid after 1 or 2 days of incubation at 37°C were subcultured onto kanamycin esculin azide agar (Lab M) to which 20 μg of vancomycin per ml was added.

179 SANTE20
Consultation sur le développement durable de la production porcine au Québec

6211-12-007
A horse was identified as *E. faecalis*, and another strain from a pheasant was identified as *E. gallinarum*.

All glycopeptide-resistant strains carried the vanA gene. The single *E. gallinarum* strain from a pheasant also possessed the vanC1 gene.

**DISCUSSION**

No glycopeptide resistance was found among enterococcal strains isolated on vancomycin-free media in Europe shortly after the introduction of avoparcin in European animal husbandry (7) or in more recently isolated North American strains (15). The VRE strains from animals described to date (1, 2, 12) have been isolated selectively on vancomycin-containing liquid or solid medium. The vancomycin selective enrichment procedures used in those studies and in the present work detect only the VanA phenotype. The vancomycin concentration of 20 μg/ml of the enrichment broths is too high to allow for the growth of strains with the low-level glycopeptide resistance phenotypes VanB and VanC. The only vanC-positive strain, an *E. gallinarum* isolate recovered from an enrichment culture of the intestinal contents of a pheasant, was probably detected because it was additionally vanA-positive. A similar strain has been described recently from a human patient taking vancomycin orally (5). As in humans, most VRE isolates identified here were *E. faecium*. Vancomycin-resistant *E. durans* strains have been described in two renal transplant patients (9), but apparently, such strains are rare in animals and humans.

Sample selection in the present study was determined by our primary aim, which was to study samples from as many different animal origins as possible. For this reason only one sample per farm or per owner was examined. The selection of the material may be biased because many samples originated from farms experiencing disease or increased mortality. However, most diseases diagnosed in the necropsied animals included in the study were not due to bacteria and virtually none were due to enterococci. For some of the animal species less frequently kept as farm or pet animals, the number of samples was too low to allow us to draw conclusions regarding the prevalence of glycopeptide resistance. Nevertheless, it can be assumed that the material examined was fairly representative of the animal exposures that humans may have.

We concluded that vanA-mediated glycopeptide resistance is widespread in enterococci from animals, at least in the species *E. faecium*. Our results confirm and expand those of Bates and colleagues (2), who found vancomycin-resistant *E. faecium* strains in 15 of 36 pigs sampled at an experimental field station and in a duck, a chicken, a turkey, a dog, a pony, and 2 pigs on a single small farm.

Our purpose to examine material from diverse origins made it difficult to obtain dependable information on the often complex and variable antibiotic use in the animals and on the farms sampled. Although pet animals do not receive antibiotics in their food, at least in some of them acquired vanA-based resistance occurs. The possibility that these animals may have acquired these strains from meat products in their feeds originating from animals fed avoparcin or from recently contaminated foods cannot be excluded. Whether feeding glycopeptide antibiotics such as avoparcin results in an increased incidence of acquired glycopeptide resistance, as has been suggested previously (1, 11), should be confirmed by carefully controlled studies. Answers to this question and investigations on the possible epidemiological relationships between animal and human glycopeptide-resistant enterococci, and notably *E. faecium*, are of utmost importance.

**ACKNOWLEDGMENT**

P.V. is indebted to the National Fund for Scientific Research for a position as a postdoctoral research fellow.

**REFERENCES**


